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TRANSACTIONS

OF THE

AMERICAN

SURGICAL ASSOCIATION

VOLUME THE FORTY-SECOND

ROYAL SOCIETY OF MEDICINE
11 JUN
1925

EDITED BY

JOHN H. JOPSON, M.D.

RECORDER OF THE ASSOCIATION

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1924

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NOTICE

This volume of TRANSACTIONS contains the papers read before the Association at the Meeting held April 17, 18 and 19, 1924.

The Association assumes no responsibility for the statements and opinions published in this volume.

OFFICERS OF THE ASSOCIATION

ELECTED APRIL, 1924

PRESIDENT

ALBERT J. OCHSNER, M.D.

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STANLEY STILLMAN, M.D.

SECRETARY

ROBERT B. GREENOUGH, M.D.,
8 Marlborough Street, Boston, Mass.

TREASURER

CHARLES H. PECK, M.D.,
30 West Fiftieth Street, New York City.

RECORDER

JOHN H. JOPSON, M.D.,
1824 Pine Street, Philadelphia, Pa.

COUNCIL

L. L. McARTHUR, M.D.
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ALBERT J. OCHSNER, M.D.
ROBERT B. GREENOUGH, M.D.
CHARLES H. PECK, M.D.
JOHN H. JOPSON, M.D.

Ex-officio.

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PRESIDENT, SECRETARY, RECORDER AND THE CHAIRMAN OF THE ADJUNCT LOCAL COMMITTEE OF ARRANGEMENTS, *ex-officio.*

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(Report by Title)

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ASTLEY P. C. ASHHURST, M.D.
LINCOLN DAVIS, M.D.

Place of next meeting:

WASHINGTON, D. C.

ADJUNCT LOCAL COMMITTEE OF ARRANGEMENTS

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HARRY H. KERR, M.D.
LIEUT.-COL. WILLIAM L. KELLER, M.C., U.S.A.

DELEGATE TO TRIENNIAL CONGRESS IN WASHINGTON

WILLIAM H. CARMALT, M.D.

LIST OF THE PRESIDENTS

OF THE

ASSOCIATION FROM THE TIME OF ITS INSTITUTION

‡ Deceased.

ELECTED

1880. ‡SAMUEL DAVID GROSS, M.D., LL.D., D.C.L. (Oxon.), LL.D. (Cantab.), LL.D. (Edin.)—Founder of the Association.
1883. ‡EDWARD MOTT MOORE, M.D., LL.D.
1884. ‡WILLIAM THOMPSON BRIGGS, M.D.
1885. ‡MOSES GUNN, A.M., M.D., LL.D.
1886. ‡HUNTER McGUIRE, M.D., LL.D.
1887. ‡D. HAYES AGNEW, M.D., LL.D.
1888. ‡DAVID WILLIAMS CHEEVER, M.D., LL.D.
1889. ‡DAVID W. YANDELL, M.D.
1890. ‡CLAUDIUS HENRY MASTIN, M.D., LL.D.
1891. ‡PHINEAS SANBORN CONNER, M.D., LL.D.
1892. ‡NICHOLAS SENN, M.D., Ph.D., LL.D.
1893. ‡JAMES EWING MEARS, A.M., M.D., LL.D.
1894. FREDERIC SHEPARD DENNIS, M.D.
1895. ‡LOUIS McLANE TIFFANY, A.M. (Cantab.), M.D.
1896. JOHN COLLINS WARREN, M.D., LL.D. (Jefferson) Hon. F.R.C.S. (Eng.).
1897. ‡THEODORE F. PREWITT, M.D.
1898. WILLIAM W. KEEN, A.M., M.D., LL.D., Hon. F.R.C.S. (Eng., Edin. and Ireland), Hon. F.A.C.S.
1899. ROBERT F. WEIR, A.M., M.D., Hon. F.R.C.S. (Eng.).
1900. ‡ROSWELL PARK, M.D.
1901. ‡DE FOREST WILLARD, A.M., M.D., Ph.D.
1902. ‡MAURICE H. RICHARDSON, A.B., M.D.
1903. ‡NATHANIEL P. DANDRIDGE, M.D.
1904. ‡GEORGE BEN JOHNSTON, M.D.
1905. ALBERT VANDER VEER, M.D.
1906. ‡DUDLEY P. ALLEN, M.D., LL.D.
1907. WILLIAM H. CARMALT, M.D.
1908. ‡C. B. G. de NANCRÈDE, A.M., M.D., LL.D.

ELECTED

1909. RUDOLPH MATAS, M.D.
1910. RICHARD H. HARTE, M.D.
1911. ‡ARPAD G. GERSTER, M.D., CHIR.D.
1912. ‡CHARLES A. POWERS, A.M., M.D.
1913. WILLIAM J. MAYO, A.M., M.D., D.SC., F.R.C.S. (EDIN.), LL.D. (TOR.).
1914. GEORGE E. ARMSTRONG, C.M., M.D.
1915. ‡ROBERT G. LE CONTE, A.B., M.D.
1916. SAMUEL J. MIXTER, S.B., M.D.
1917. THOMAS W. HUNTINGTON, A.B., M.D., LL.D.
1918. LEWIS S. PILCHER, A.M., M.D., LL.D.
1919. GEORGE E. BREWER, M.D.
1920. ‡JOHN B. ROBERTS, M.D.
1921. JOHN M. T. FINNEY, M.D.
1922. L. L. MCARTHUR, M.D.
1923. GEORGE W. CRILE, M.D.
1924. ALBERT J. OCHSNER, M.D.

FELLOWS

OF THE

AMERICAN SURGICAL ASSOCIATION

APRIL, 1924

* Denotes Original Fellows.
† Denotes Senior Fellows.
‡ Denotes Deceased Fellows.

ELECTED

†1890. ABBE, ROBERT, M.D., 40 W. Fifty-ninth Street, New York. Senior Surgeon to St. Luke's Hospital. *Vice-President*, 1901. *Council*, 1908.

1902. ANDREWS, E. WYLLYS, A.M., M.D., 122 S. Michigan Avenue, Chicago, Ill. Professor of Surgery, Northwestern University Medical School; Surgeon to St. Luke's Hospital and Michael Reese Hospital; Surgeon-in-Chief to Cook County Hospital; Consulting Surgeon to the Provident Hospital. *Vice-President*, 1923.

1916. ARCHIBALD, EDWARD WILLIAM, B.A., M.D., C.M., 52 Westmount Boulevard, Montreal, Canada. Professor of Surgery and Director of the Surgical Department, McGill University; Surgeon, Royal Victoria Hospital; Associate Surgeon, Children's Memorial Hospital.

†1901. ARMSTRONG, GEORGE E., M.D., C.M., C.M.G., LL.D. (Queens), D.Sc. (Liverpool), M.Ch. (Dublin), 320 Mountain Street, Montreal. Emeritus Professor of Surgery and of Clinical Surgery, McGill University; Consulting Surgeon to the Royal Victoria Hospital, the Montreal General Hospital, the Western Hospital and to the Protestant Hospital for the Insane, Verdun; Colonel, C.A.M.C. *President*, 1914.

ELECTED

1913. Ashhurst, Astley Paston Cooper, A.B., M.D., N. E. Corner Sixteenth and Spruce Streets, Philadelphia. Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to the Episcopal Hospital; Surgeon to the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases; Colonel, Medical Reserve Corps, U. S. Army.

1922. Bailey, Fred Warren, B.Sc., M.D., 611 Metropolitan Building, St. Louis, Mo. Associate Professor of Surgery, St. Louis University School of Medicine; Surgeon to St. John's Hospital; Visiting Surgeon, St. Louis City Hospital; Surgeon and Chief of Staff, Shriners' Hospital.

1920. Balch, Franklin G., A.M., M.D., F.A.C.S., 279 Clarendon Street, Boston, Mass. Chief of East Surgical Service, Massachusetts General Hospital; Surgeon at the Faulkner Hospital; Associate in Surgery, Graduate Courses in Medicine, Harvard Medical School.

1917. Balfour, Donald Church, M.B., M.D., Rochester, Minn. Professor of Surgery, University of Minnesota; Chief of Surgery, Mayo Foundation; Surgeon to St. Mary's Hospital, Rochester. *Vice-President*, 1922.

1906. Barrow, David, M.D., 190 N. Upper Street, Lexington, Ky. Surgeon to St. Joseph and Good Samaritan Hospitals; recently Lieut.-Col., M.C.; Commanding Officer, Base Hospital 40.

†1882. Barton, James M., A.M., M.D., 32 North New Hampshire Avenue, Atlantic City, N. J. (Retired.) Formerly Professor of Clinical Surgery, Jefferson Medical College; and Surgeon to the Philadelphia and German (now Lankenau) Hospitals.

1924. Beer, Edwin, A.B., M.D., 11 East Forty-eighth Street, New York City. Attending Surgeon, Bellevue Hospital, 4th Division, and at Mt. Sinai Hospital.

1915. Besley, Frederic A., M.D., F.A.C.S., Sheridan Road, Waukegan, Ill. Professor of Surgery, Northwestern University Medical School; Attending Surgeon, Victory Memorial Hospital, Waukegan, and Cook County Hospital, Chicago, Ill.

1900. Bevan, Arthur Dean, M.D., 122 S. Michigan Boulevard, Chicago. Professor and Head of the Department of Surgery, Rush Medical College, University of Chicago; Surgeon to Presbyterian Hospital.

ELECTED

†1901. BINNIE, JOHN FAIRBAIRN, M.A., M.B., C.M. (Aberdeen), 917 Rialto Building, Kansas City, Mo. *Vice-President*, 1907 and 1919.

1918. BLAIR, VILRAY PAPIN, M.D., 4654 Pershing Avenue., St. Louis, Mo. Professor of Oral Surgery, Washington University Dental School; Associate Professor of Clinical Surgery, Washington University Medical School; Visiting Surgeon, St. Luke's Hospital; Associate Surgeon to St. Louis Maternity and Mullanphy Hospital, St. Louis Children's Hospital, and Barnes Hospital.

†1906. BLAKE, JOHN BAPST, A.M., M.D., 311 Beacon Street, Boston, Mass. Assistant Professor in Surgery, Harvard Medical School; Surgeon-in-Chief to the Boston City Hospital; Consulting Surgeon to Long Island Hospital; Consulting Surgeon to Boston Insane Hospital, and to the Reformatory for Women, Framingham, Mass.

†1902. BLAKE, JOSEPH A., A.B., PH. B., M.A. (Hon.), M.D., F.A.C.S., 116 East Fifty-third Street, New York. International Surgical Society; American Association of Anatomists; Corresponding Member, French Surgical Society; New York Academy of Medicine. Surgical Director, Reconstruction Hospital; Consulting Surgeon, Roosevelt, St. Luke's, Lincoln, St. John's (Yonkers), Tarrytown and Dobbs Ferry Hospitals.

1901. BLOODGOOD, JOSEPH COLT, B.S. (Wisc., 1888), M.D. (Penna., 1891); *Vice-President*, office, 904 N. Charles Street, Baltimore, Md.; residence, 18 Midvale Road, Roland Park, Md. Associate Professor of Clinical Surgery, Johns Hopkins University; Surgeon to St. Agnes's Hospital, Baltimore.

1913. BOTTOMLEY, JOHN T., A.B., LL.D., M.D., 165 Beacon Street, Boston. Surgeon-in-Chief, Carney Hospital; Associate in Surgery, Course for Graduates, Harvard Medical School; Consulting Surgeon to sundry Hospitals.

†1890. BRADFORD, EDWARD HICKLING, A.B., M.D., 253 Newbury Street, Boston. Former Dean and Professor Emeritus of Orthopedic Surgery, Harvard University Medical School; Consulting Surgeon, Children's Hospital and Boston City Hospital; Member of International Surgical Association, American Orthopedic Association, American College of Surgeons, Massachusetts Medical Society.

ELECTED

†1900. BREWER, GEORGE EMERSON, M.D., LL.D., F.A.C.S., F.R.C.S. (Ireland), 19 East Sixty-fifth Street, New York City. Emeritus Professor of Surgery in College of Physicians and Surgeons, Columbia University; Consulting Surgeon to the Presbyterian Hospital, Roosevelt Hospital, City Hospital, Woman's Hospital, St. Vincent's Hospital, Knickerbocker Hospital, Fifth Avenue Hospital, Manhattan Eye, Ear, Nose and Throat Hospital, Ophthalmic and Aural Institute, St. Bartholomew's Hospital and Clinic, and House of the Holy Comforter, New York; the Flushing Hospital, Perth Amboy Hospital, the Muhlenberg Hospital of Plainfield, N. J., and Christ Hospital, Jersey City. *Vice-President*, 1913; *President*, 1919. Member of Council, 1920–23.

1917. BREWSTER, GEORGE W. W., A.B., M.D., F.A.C.S., 213 Beacon Street, Boston, Mass. Associate in Surgery, Harvard Graduate School; Visiting Surgeon, Massachusetts General Hospital; Consulting Surgeon to Frost Hospital, Chelsea, Mass.; Leonard Morse Hospital, Natick, Mass.; Milford Hospital, Milford, Mass.; Brockton Hospital, Brockton, Mass.

1915. BRINSMADE, W. B., A.B., M.D., 172 Clinton St., Brooklyn, N. Y. Consulting Surgeon to St. Christopher's Hospital; Senior Surgeon, Brooklyn Hospital; Commander, U.S.N.R.F., M.C.

1911. BRUCE, HERBERT A., M.D. (Toronto Univ.), L.R.C.P. (London), F.R.C.S. (England), 64 Bloor Street East, Toronto, Canada. Professor of Clinical Surgery, University of Toronto; Consulting Surgeon to the Toronto General Hospital; Colonel, Canadian Army Medical Corps and Royal Army Medical Corps; Consulting Surgeon to the British Armies in France, 1917–18.

1911. BUCHANAN, J. J., A.M., M.D., PH.D., 1409 N. Highland Avenue, Pittsburgh, Pa. Professor of Surgery, University of Pittsburgh; Surgeon to Mercy Hospital.

†1900. BUNTS, FRANK E., M.D., Euclid Avenue and Ninety-third Street, Cleveland, Ohio. Professor of Principles of Surgery and Clinical Surgery in Medical College of Western Reserve University; Visiting Surgeon and Chief of Staff to St. Vincent's Charity Hospital.

1924. CABOT, HUGH, A.B., M.D., University Hospital, Ann Arbor, Mich. Professor of Surgery, University of Michigan; Surgeon-in-Chief, University Hospital.

ELECTED

1921. Campbell, William Francis, A.B., M.D., F.A.C.S., 1835 East New York Avenue, Brooklyn, N. Y. Emeritus Professor of Surgery, Long Island College Hospital; Surgeon-in-Chief to the Trinity Hospital; Consulting Surgeon to the Methodist Episcopal, East New York and Brownsville, Long Island College, Coney Island, Swedish, Jamaica, and Zion Hospitals; Clinical Director of the Brooklyn Cancer Research Hospital.

†1885. Carmalt, William H., A.M., M.D., 261 St. Ronan Street, New Haven, Conn. Emeritus Professor of Surgery in Yale University; Vice-President and Consulting Surgeon to the New Haven Hospital. *President*, 1907; *Vice-President*, 1895; *Member of Council*, 1908.

1909. Carrel, Alexis, L.B., Sc.B., M.D., Rockefeller Institute for Medical Research, Sixty-sixth Street and Avenue A, New York, N. Y. *Vice-President*, 1919.

†1896. Carson, Norman Bruce, M.D., 303 Humboldt Building St. Louis, Mo. Surgeon to St. Louis and Mullanphy Hospitals and to the St. Vincent Insane Asylum, etc. *Vice-President*, 1903.

1922. Cheever, David, A.B., M.D., Peter Bent Brigham Hospital, Boston, Mass. Associate Professor of Surgery, Harvard Medical School; Surgeon to the Peter Bent Brigham Hospital.

1920. Clinton, Marshall, M.D., 556 Franklin Street, Buffalo, New York. Associate Professor of Surgery, University of Buffalo, Department of Medicine; Professor of Oral Surgery, University of Buffalo, Dental Department; Attending Surgeon, Buffalo General, Erie County, and Buffalo City Hospitals.

1920. Clopton, Malvern Bryan, M.D., 405 Humboldt Building, St. Louis, Mo. Associate in Surgery, Washington University Medical School; Visiting Surgeon, St. Luke's Hospital; Assistant Surgeon, Barnes Hospital; Associate Surgeon, St. Louis Children's Hospital.

1916. Codman, Ernest Amory, A.B., M.D., 227 Beacon Street, Boston, Mass.

†1898. Coley, William B., M.D., The Medical Chambers, 114 East Fifty-fourth Street, New York. Attending Surgeon to the Ruptured and Crippled and the Memorial Hospitals; Consulting Surgeon to the Fifth Avenue, the Mary McClellan (Cambridge, N. Y.), and the Sharon (Sharon, Conn.)

ELECTED

Hospitals; Professor of Clinical Cancer Research at Memorial Hospital, Cornell University Medical College.

1922. Connors, John Fox, A.M., M.D., 59 East Fifty-fourth Street, New York City. Surgical Director, Harlem Hospital, New York City; Consulting Surgeon, St. Mary's Hospital, Waterbury, Conn.

1914. Cotton, Frederick J., A.B., M.D., A.M., 520 Commonwealth Avenue, Boston, Mass. Visiting Surgeon to the Boston City Hospital; Surgeon-in-Chief, Beth Israel Hospital; Associate in Surgery, Harvard Medical School; Consultant in Surgery, United States Public Health Service, Northeastern District; also Consultant in Surgery, New England Hospital for Women and Children; Consultant in Surgery, Peterboro Hospital.

†1905. Crile, George W., M.D., *Member of Council*, Euclid Avenue and Ninety-third Street, Cleveland, Ohio. Surgeon, Cleveland Clinic Hospital. *President*, 1923.

1906. Cushing, Harvey, A.M., M.D., Sc.D., LL.D., F.R.C.S., 721 Huntington Avenue, Boston. Professor of Surgery, Harvard University.

†1896. Cushing, Hayward Warren, A.B., M.D., 70 Commonwealth Avenue, Boston, Mass. Consulting Surgeon, Boston City Hospital.

†1897. Da Costa, John Chalmers, M.D., 2045 Walnut Street, Philadelphia. Samuel D. Gross Professor of Surgery, Jefferson Medical College; Surgeon to the Hospital of the Jefferson Medical College; Consulting Surgeon to the Philadelphia General Hospital, Saint Joseph's Hospital and Misericordia Hospital.

1917. Darrach, William, A.B., M.A., M.D., 437 West Fifty-ninth Street, New York City. Associate Professor of Surgery and Dean of the Medical Faculty, College of Physicians and Surgeons, Columbia University; Consulting Surgeon, Presbyterian Hospital, New York City; Greenwich Hospital, Greenwich, Connecticut; St. John's Hospital, Yonkers, N.Y., and Memorial Hospital, Morristown, N. J.

1923. David, Vernon C., A.B., M.D., 25 East Washington Street, (Room 1927), Chicago, Ill. Assistant Professor of Surgery, Rush Medical College; Associate Attending Staff, Surgery, Presbyterian Hospital; Attending Staff, Surgery, Cook County Hospital; Senior Attending Staff, Surgery, Evanston Hospital.

ELECTED

1916. Davis, Carl, B., A.B., M.D., 122 South Michigan Boulevard, Chicago, Ill. Associate Professor of Clinical Surgery, Rush Medical College; Associate Surgeon, Presbyterian Hospital, Chicago.

1917. Davis, John Staige, Ph.B., M.D., F.A.C.S., Severn Apartments, Baltimore, Md. Associate Professor of Clinical Surgery, Johns Hopkins University; Visiting and Plastic Surgeon, Union Memorial Hospital, Hospital for the Women of Maryland, Children's Hospital School; Assistant Visiting Surgeon and Dispensary Surgeon, Johns Hopkins Hospital.

1919. Davis, Lincoln, A.B., M.D., 279 Beacon Street, Boston, Mass. Associate in Surgery, Harvard Graduate School of Medicine; Visiting Surgeon, Massachusetts General Hospital; Consulting Surgeon, Heywood Memorial Hospital, Gardner; Milford Hospital, Milford.

1912. Deaver, Harry C., M.D., 1701 Spruce Street, Philadelphia. Surgeon-in-Chief to Kensington Hospital for Women; Surgeon to the Episcopal Hospital and to the Children's Hospital of the Mary Drexel Home.

1892. Deaver, John B., M.D., Sc.D., LL.D., 1634 Walnut Street, Philadelphia. Emeritus John Rhea Barton Professor of Surgery, University of Pennsylvania, Philadelphia; Surgeon-in-Chief to the Lankenau Hospital, Philadelphia.

1910. Delatour, H. Beeckman, M.D., 73 Eighth Avenue, Brooklyn, N. Y. Attending Surgeon, St. John's and Norwegian Hospitals; Consulting Surgeon, Kingston Avenue Hospital, Brooklyn; St. Mary's Hospital, Jamaica; Northport Hospital, and South Side Hospital, Bay Shore.

†1882. Dennis, Frederic Shepard, M.D., F.R.C.S. (Eng.), 62 East Fifty-fifth Street, New York. Professor Emeritus of Clinical Surgery, Cornell University, New York City; Consulting Surgeon to Bellevue and St. Vincent's Hospitals; St. Joseph's Hospital, Yonkers, and Montefiore Home, New York City; Litchfield County Hospital, Winsted, Conn. *President*, 1894; *Vice-President*, 1887; *Member of Council*, 1889.

1924. Douglass, John, Sc.B., M.D., 568 Park Avenue, New York City. Clinical Professor of Surgery, New York University and Bellevue Medical College; Attending Surgeon, Bellevue Hospital; Associate Surgeon, St. Luke's Hospital; Surgical Director, Knickerbocker Hospital.

1910. Dowd, Charles N., A.M., M.D., Sc.D., 138 West Fifty-eighth Street, New York, N. Y. Professor of Clinical

ELECTED

Surgery, Columbia University; Consulting Surgeon, Roosevelt Hospital, Memorial Hospital, St. Mary's Free Hospital for Children, New York, Richmond Memorial Hospital, and Lulu Thorley Home for Crippled Children. *Member of Council*, 1918.

1914. DOWNES, WILLIAM A., M.D., 424 Park Avenue, New York City. Professor of Clinical Surgery, Columbia University, College of Physicians and Surgeons; Attending Surgeon, St. Luke's and Babies' Hospitals; Consulting Surgeon, Memorial, Stamford Hospital, Stamford, Conn., Manhattan State Hospital, Hospital for Ruptured and Crippled, Infirmary for Women and Children, New York, and United Hospital, Portchester, N. Y.

1924. DRENNEN, EARLE, A.B., M.D., 519 American Trust Building, Birmingham, Alabama. Attending Surgeon, Hillman Hospital, St. Vincent's Hospital and Children's Hospital.

1919. EASTMAN, JOSEPH RILUS, A.M., B.Sc., M.D., F.A.C.S., 331 North Delaware Street, Indianapolis, Ind. Professor of Surgery, Indiana University School of Medicine; Visiting Surgeon to City Hospital.

1901. ELIOT, ELLSWORTH, JR., M.D., 34 East Sixty-seventh Street, New York. Surgeon to the Polyclinic Hospital; Consulting Surgeon to the Grosveneur Hospital, N. Y.; Lawrence Hospital, Bruneville, N. Y.; Nasson Hospital, Mineola, L. I.; White Plains Hospital, White Plains, N. Y.; Hackensack Hospital, Hackensack, N. J.; Mountainside Hospital, Montclair, N. J.; Skin and Cancer Hospital, New York and Northeastern Dispensary, New York. *Vice-President*, 1922.

†1893. ELLIOT, JOHN WHEELOCK, A.M., M.D. (Harvard), 124 Beacon Street, Boston. Surgeon to Massachusetts General Hospital, 1893–1907; Clinical Instructor in Gynecology, 1888–9, and Lecturer on Surgery, 1900–5, Harvard Medical School; Chairman, N. E. Surgical Dressings Committee, 1917.

1911. ELTING, ARTHUR W., A.B., M.D., LL.D., 119 Washington Avenue, Albany, N. Y. Professor of Surgery in the Albany Medical College; Surgeon to the Albany Hospital and to the Child's Hospital, Albany, N. Y.

†1896. ESTES, WILLIAM LAWRENCE, A.M., M.D., Bethlehem, Pa. Emeritus Surgeon-in-Chief of St. Luke's Hospital.

ELECTED

†1898. Eve, Duncan, A.M., M.D., Eve Building, Nashville, Tenn. Head of the Department and Professor of Surgery and Clinical Surgery, Medical Department, Vanderbilt University; Chief Surgeon of the Louisville & Nashville Railroad; Chief Surgeon of the N. C. & St. Louis Railroad; Attending Surgeon to St. Thomas' Hospital; Consulting Surgeon to Nashville City and Woman's Hospitals.

†1899. Finney, John M. T., M.D., *Member of Council*, 1300 Eutaw Place, Baltimore, Md. Professor of Clinical Surgery, Johns Hopkins University; Surgeon-in-Chief to Johns Hopkins Hospital. *President*, 1921.

1921. Fisher, William A., M.D., 715 Park Avenue, Baltimore, Md. Associate in Clinical Surgery, Johns Hopkins University; Assistant Visiting Surgeon, Johns Hopkins Hospital; Visiting Surgeon, Union Memorial Hospital, Church Home and Infirmary, Hospital for the Women of Maryland, Children's Hospital School.

1904. Frazier, Charles Harrison, M.D., Sc.D., 3600 Walnut Street, Philadelphia, Pa. John Rhea Barton Professor of Surgery, University of Pennsylvania; Surgeon-in-Chief to the University Hospital.

†1898. Freeman, Leonard, B.S., M.A., M.D., 424 Metropolitan Building, Denver, Col. Professor of Surgery, Medical Department, University of Colorado; Surgeon to St. Joseph's Hospital; Consulting Surgeon to Children's, St. Luke's, Beth Israel and National Jewish Hospitals, and to the Denver General Hospital, Denver, Col. *Vice-President*, 1908.

1910. Gage, Homer, A.M., M.D., 72 Pearl Street, Worcester, Mass. Surgeon-in-Chief to Memorial Hospital; Consulting Surgeon to Worcester City and St. Vincent's Hospitals. *Vice-President*, 1923.

1922. Gallie, William E., M.D., F.A.C.S., F.R.C.S. (Eng.), 143 College Street, Toronto, Canada. Hunterian Professor, Royal College of Surgeons of England; Associate Professor of Surgery, University of Toronto; Surgeon-inChief, Hospital for Sick Children, Toronto.

1919. Gaub, Otto Carl, M.D., F.A.C.S. Hotel Schenley, Pittsburgh, Pa. Surgeon to Allegheny General Hospital and to Columbia Hospital.

ELECTED

†1882. Gay, George Washington, A.M., M.D., Chestnut Hill, Mass. Senior Surgeon to the Boston City Hospital. *Member of Council*, 1882.

1906. Gibbon, John H., M.D., 1608 Spruce Street, Philadelphia. Professor of Surgery, Jefferson Medical College and Surgeon to the Jefferson College Hospital; Surgeon to the Pennsylvania Hospital and Consulting Surgeon to the Bryn Mawr Hospital. *Secretary*, 1915–1922.

1906. Gibson, C. L., M.D., 72 East Fifty-fourth Street, New York, N. Y. Professor of Surgery, Cornell University Medical College; Attending Surgeon to the First Surgical (Cornell) Division of the New York Hospital. Membre Associé-étranger Société Nationale de Chirurgie de Paris; Consulting Surgeon to St. Luke's, City, and General Memorial Hospitals; New York State Hospital for Care of Crippled and Deformed Children; South Side Hospital, Bay Shore; Vassar Brothers Hospital, Poughkeepsie. *Vice-President*, 1915.

1920. Graham, Evarts A., A.B., M.D., Euclid Avenue and Kingshighway, St. Louis, Mo. Professor of Surgery, Washington University Medical School, St. Louis, Missouri; Surgeon-in-Chief to Barnes Hospital and St. Louis Children's Hospital.

1911. Greenough, Robert B., A.B. (Harvard), M.D. (Harvard), *Secretary*, 8 Marlborough Street, Boston, Mass. Assistant Professor of Surgery, Harvard Medical School; Visiting Surgeon, Massachusetts General Hospital; Director of Cancer Commission, Harvard University.

1922. Guerry, Le Grand, M.D., F.A.C.S., D.C.L., Columbia, S. C. Surgeon to Columbia Hospital, Columbia, S. C.

1924. Guthrie, Donald, Ph.B., M.D., Sayre, Pa. Surgeon-in-Chief, Robert Packer Hospital.

1916. Haggard, William D., M.D., F.A.C.S., 706 Church Street, Nashville, Tenn. Professor of Surgery, Vanderbilt University; Surgeon to St. Thomas' Hospital.

1909. Halstead, Albert E., M.D., 30 N. Michigan Avenue, Chicago, Ill. Professor of Surgery, School of Medicine, University of Illinois; Attending Surgeon to the St. Luke's Hospital; Consulting Surgeon to the Illinois Charitable Eye and Ear Infirmary.

ELECTED

1909. HAMANN, CARL A., M.D., 416 Osborn Building., Cleveland, Ohio. Professor of Applied Anatomy and Clinical Surgery, Western Reserve University; Visiting Surgeon to Charity and City Hospitals.

1900. HARRIS, MALCOLM L., M.D., 25 E. Washington Street, Chicago, Ill. Professor of Surgery, Chicago Policlinic; Attending Surgeon, Alexian Brothers' Hospital, Henrotin Hospital, Passavant Hospital.

†1895. HARTE, RICHARD H., M.D., C.M.G., 1503 Spruce Street, Philadelphia. Emeritus Surgeon to the Pennsylvania Hospital and Consulting Surgeon to St. Mary's, Memorial, Abington, and Bryn Mawr Hospitals; Colonel M.R.C., U.S.A; formerly Director Base Hospital No. 10, U.S.A., and later O. C. No. 16, General Hospital, B.E.F. *President*, 1910.

1916. HARTWELL, JOHN A., PH.B., M.D., 27 East Sixty-third Street, New York. Associate Professor of Surgery and Professor of Clinical Surgery, Cornell University Medical College; Director of Surgery, Cornell University Division, Bellevue Hospital; Consulting Surgeon to Memorial Hospital, Reconstruction Hospital, New York Infirmary for Women and Children, Lincoln Hospital, Lawrence Hospital, Bronxville, N. Y., and United Hospital, Portchester, N. Y.

1922. HEUER, GEORGE J., M.D., Cincinnati General Hospital, Cincinnati, Ohio. Professor of Surgery, University of Cincinnati College of Medicine; Surgical Director of the Cincinnati General Hospital.

1922. HITZROT, JAMES MORLEY, A.B., M.D., 126 East Thirty-seventh Street, New York City. Professor of Clinical Surgery, Cornell Medical School; Associate Surgeon, New York Hospital.

1918. HODGE, EDWARD BLANCHARD, A.B., M.D., 2019 Spruce Street, Philadelphia, Pa. Surgeon to Presbyterian and Children's Hospitals; Associate Surgeon to Pennsylvania Hospital; Associate Surgeon to Widener Memorial School.

1923. HOMANS, JOHN, A.M., M.D., Peter Bent Brigham Hospital, 721 Huntington Avenue, Boston, Mass. Instructor in Surgery, Harvard Medical School; Surgeon to the Peter Bent Brigham Hospital.

1924. HORSLEY, J. SHELTON, M.D., St. Elizabeth's Hospital, Richmond, Va. Attending Surgeon, St. Elizabeth's Hospital.

ELECTED

1909. Hotchkiss, Lucius Wales, A.B., M.D., San Marcos Building, Santa Barbara, Cal. Professor of Clinical Surgery, Columbia University; Consulting Surgeon to Bellevue, Knickerbocker, Flushing, Roosevelt, and Greenwich Hospitals, N. Y.

1918. Hubbard, Joshua C., M.D., 86 Bay State Road, Boston, Mass. Chief of Third Surgical Service, Boston City Hospital; Surgeon, Newton Hospital; Consulting Surgeon, Norwood and Leonard Morse Hospitals.

†1901. Huntington, Thomas W., A.B., M.D., LL.D., 960 Mills Building, San Francisco, California. Professor of Clinical and Operative Surgery (Emeritus), College of Medicine, University of California; Consulting Surgeon, St. Luke's Hospital, San Francisco; Chief Surgeon, Western Pacific R. R. *Vice-President*, 1906; *President*, 1917; *Member of Council*, 1918–20.

1919. Hupp, Frank Le Moyne, A.M., M.D., 61 Fourteenth Street, Wheeling, W. Va. Attending Surgeon, Ohio Valley General Hospital; Consulting Surgeon, Reynolds Memorial Hospital, Glendale.

†1909. Hutchinson, James P., M.D., Media, Pa.

1913. Hutchison, James Alexander, C.B.E., M.D., (McGill), L.R.C.P. and S. (Edin.), 354 MacKay Street, Montreal. Emeritus Professor of Surgery and Clinical Surgery, McGill University; Consulting Surgeon, Montreal General and Royal Victoria Hospitals.

1913. Jackson, Jabez N., A.M., M.D., 425 Argyle Building, Kansas City, Mo. Surgeon, Christian Church and Kansas City General Hospitals.

†1905. Johnson, Robert W., A.B., M.D., 101 W. Franklin Street, Baltimore, Md. Professor of Principles and Practice of Surgery, Baltimore Medical College; Surgeon to Maryland General Hospital, Church Home and Infirmary, and St. Agnes' Hospital. Retired.

†1901. Jonas, A. F., M.D., 612 Omaha Loan Building, Omaha, Neb. Head of the Department in Surgery, College of Medicine, University of Nebraska (formerly Omaha Medical College); Surgeon-in-Chief, Omaha Methodist Episcopal Hospital; Captain, M.C. *Vice-President*, 1906.

ELECTED

1917. JONES, DANIEL FISKE, A.B., M.D., 195 Beacon Street, Boston. Mass. Associate in Surgery, Courses for Graduates, H.M.S.; Visiting Surgeon to Massachusetts General Hospital, and M.E. Deaconess' Hospital; Consulting Surgeon to the New England Hospital for Women and Children, Brockton City Hospital, Union Hospital of Fall River, Sturdy Hospital of Attleboro and the Addison Gilbert Hospital of Gloucester, Mass.

1910. JOPSON, JOHN HOWARD, M.D., *Recorder*, 1824 Pine Street, Philadelphia, Pa. Professor of Surgery, the Graduate School of Medicine, University of Pennsylvania; Surgeon to Presbyterian Hospital and Children's Hospital; Consulting Surgeon to Norristown State Hospital and Philadelphia Home for Incurables.

1914. JUDD, EDWARD STARR, M.D., Rochester, Minn., Surgeon to Mayo Clinic.

†1899. KAMMERER, FREDERIC, M.D., Lenox Hill Hospital, New York City. Professor of Clinical Surgery, Columbia University; Consulting Surgeon to the Lenox Hill Hospital, and to St. Francis Hospital, New York.

1913. KANAVEL, ALLEN B., A.B., M.D., 30 North Michigan Avenue, Chicago, Ill. Professor of Surgery, Northwestern University Medical School; Attending Surgeon to Wesley Memorial and Cook County Hospitals.

*†1880. KEEN, WILLIAM WILLIAMS, A.M., M.D., LL.D., Hon. F.R.C.S. (Eng., Edin. and Ireland), Hon. Ph.D. (Upsala), Hon. ScD. (Jeff. and Harv.), Hon. F.A.C.S., The Touraine, 1520 Spruce Street, Philadelphia. Emeritus Professor of Surgery in the Jefferson Medical College; Consulting Surgeon to the Jefferson Medical College Hospital and to the Woman's and St. Agnes' Hospitals; Membre Correspondant Etranger de la Société de Chirurgie de Paris and of the Académie de Médecine de Paris; Membre Honoraire de la Société Belge de Chirurgie; Honorary Member, Clinical Society of London; Royal Society of Medicine, London; Doctor honoris causa of the University of Paris; Bigelow Medal of the Boston Surgical Society, etc. *President*, 1898; *Vice-President*, 1892; *Member of Council*, 1899–1901.

1923. KELLER, WILLIAM L., M.D., Walter Reed General Hospital, Washington, D. C. Colonel, M. C., U. S. A.; Professor of

ELECTED

Military Surgery, Army Medical School, Army Medical Center, Washington, D. C.; Chief of the Surgical Service, Walter Reed General Hospital.

1924. KERR, HARRY HYLAND, M.D., C.M. (McGill), 1744 N. Street, N.W., Washington, D.C. Professor of Neurosurgery and Clinical Professor of Surgery, George Washington University; Attending Surgeon, Garfield Memorial and Children's Hospitals; Consulting Surgeon, Providence and Columbia Hospitals; formerly Attending Surgeon, Freedman's Hospital, Washington, D. C.; Lieut.-Colonel, M.R.C.

1916. LAW, ARTHUR AYER, M.D., F.A.C.S., 420 Syndicate Building, Minneapolis, Minn. Associate Professor of Surgery, University of Minnesota Medical School, and Mayo Foundation; Surgeon to the Northwestern Hospital; Surgeon to Glen-Lake Sanatorium; Surgeon to and Assistant Chief of the University Hospital; Lieut.-Col., M.C. (discharged), Commanding University of Minnesota Base Hospital No. 26 in A.E.F.

‡1901. LE CONTE, ROBERT G., A.B., M.D., 2000 Spruce Street, Philadelphia. Chief of Surgical Service, Pennsylvania Hospital; Consulting Surgeon to the Germantown and the Bryn Mawr Hospitals; Advisory Surgeon to Pennsylvania Railroad. *President*, 1915; *Member of Council*, 1916–18.

1919. LEE, BURTON JAMES, PH.B., M.D., F.R.C.S., 128 East Seventy-third Street, New York. Clinical Professor of Surgery, Cornell Medical School; Associate Attending Surgeon, New York Hospital; Consulting Surgeon, Sharon (Connecticut) Hospital; Attending Surgeon, Memorial Hospital.

1923. LEE, WALTER ESTELL, M.D., F.A.C.S., 905 Pine Street, Philadelphia, Pa. Associate Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Pennsylvania, Germantown, Bryn Mawr and Children's Hospitals and the Associated Medical Clinics.

1912. LEWIS, DEAN D., M.D., 122 S. Michigan Ave., Chicago, Ill. Professor of Surgery, Rush Medical College; Attending Surgeon to the Presbyterian Hospital, Chicago; Consulting Surgeon to the Annie Durand Hospital for Contagious Diseases.

1910. LILIENTHAL, HOWARD, A.B., M.D., 52 East Eighty-second Street, New York. Consulting Surgeon, Mount Sinai

ELECTED

Hospital, Bellevue Hospital, Hospital for Joint Diseases, and New York Infirmary for Women and Children; Professor of Clinical Surgery, Cornell Medical College; formerly Lieut.-Col., M.C., U. S. A.

1908. Lothrop, Howard Augustus, A.M., M.D., 101 Beacon Street, Boston, Mass. Assistant Professor of Surgery, Harvard Medical School; Surgeon-in-Chief, Boston City Hospital.

1916. Lower, William E., M.D., Euclid Avenue and Ninety-third Street, Cleveland, Ohio. Associate Professor of Genito-urinary Surgery, School of Medicine, Western Reserve University; Surgeon, Cleveland Clinic Hospital; Associate Surgeon in charge of Genito-urinary Surgery, Lakeside Hospital; Visiting Surgeon, Lutheran Hospital.

1910. Lund, Fred Bates, M.D., 527 Beacon Street, Boston, Mass. Surgeon-in-Chief, Boston City Hospital; Consulting Surgeon, Burbank Hospital, Fitchburg, Newton Hospital, Newton, Choate Memorial Hospital, Woburn, Mass; lately Lieut.-Col., M.C., U. S. A.

1917. Lyle, Henry H. M., M.D., 117 East Fifty-sixth Street, New York City. Professor of Clinical Surgery, College of Physicians and Surgeons, Columbia University; Attending Surgeon, St. Luke's Hospital.

†1901. McArthur, L. L., M.D., F.A.C.S., *Member of Council*, 122 S. Michigan Boulevard, Chicago, Ill. Assistant Professor of Clinical Surgery, Rush Medical College; Senior Surgeon to St. Luke's and Michael Reese Hospitals. *President*, 1922.

1923. McGuire, Edgar R., M.D., F.A.C.S., 622 Delaware Avenue, Buffalo, N. Y. Professor of Surgery, University of Buffalo; Surgeon, Buffalo General Hospital; Surgeon-in-Chief, Buffalo City Hospital.

1917. McGuire, Stuart, M.D., LL.D., 1000 West Grace Street, Richmond, Va. President of Medical College of Virginia; Surgeon to St. Luke's Hospital; Consulting Surgeon to Memorial Hospital.

‡1903. McMurtry, Lewis S., A.M., M.D., LL.D., 542 Atherton Building, Louisville, Ky. Professor of Gynecology and Abdominal Surgery in the Medical Department, University of Louisville; Surgeon to the Louisville City Hospital.

1918. McWilliams, Clarence A., M.A., M.D., 19 East Sixty-fifth Street, New York, N. Y. Associate Surgeon, Fifth Avenue

ELECTED

and New York Polyclinic Hospitals; Attending Surgeon, New York Skin and Cancer Hospital.

‡1904. MacLaren, Archibald, M.D., B.S., 914 Lowry Building, St. Paul, Minn. Professor of Clinical Surgery, University of Minnesota. *Vice-President*, 1915. *Recorder*, 1910–1914.

1914. MacLaren, Murray, B.A., M.B.C.M., M.D., M.R.C.S. (Eng.), C.M.G., C.O.A. (Commander, Order of Avis), LL.D., M.P. (Member of Parliament), 75 Coburg Street, St. John's, N.B., Canada. Surgeon to the General Public Hospital; Colonel, C.A.M.C.

1920. Maes, Urban, M.D., 1671 Octavia Street, New Orleans, Louisiana. Professor of Clinical and Operative Surgery, Tulane University of Louisiana School of Medicine; Senior Visiting Surgeon, Charity Hospital; Senior Associate in Surgery, Touro Infirmary.

†1898. Martin, Edward, A.M., M.D., LL.D., D.Sc., Media, Pa. Professor of Surgical Physiology, University of Pennsylvania; Consulting Surgeon to State Hospitals, Norristown and Wernersville, Pa.; Colonel, M.R.C., U. S. A.

1915. Martin, Walton, PhB., M. D., 230 East Forty-ninth Street, New York. Professor of Clinical Surgery, Columbia University; Attending Surgeon, St. Luke's Hospital; Consulting Surgeon, White Plains Hospital and Loomis Sanatorium.

†1895. Mastin, William McDowell, M.D., LL.D., Northeast corner of Joachim and Conti Streets, Mobile, Ala. Surgeon to the Mobile City Hospital and Providence Infirmary.

1895. Matas, Rudolph, M.D., LL.D., 2255 St. Charles Avenue, New Orleans, Louisiana. Professor of Surgery, Tulane University of Louisiana, School of Medicine; Professor (Emeritus), Graduate Medical School, Tulane University; Chief, Surgical Division, Touro Infirmary; Senior Visiting Surgeon, Charity Hospital; Consulting Surgeon, Eye, Ear, Nose and Throat Hospital; Major, Reserve Corps, U. S. Army. *President*, 1909; *Vice-President*, 1901.

1918. Mathews, Francis Stuart, B.S., M.D., F.A.C.S., 49 West Fifty-second Street, New York. Professor of Clinical Surgery, Columbia University; Surgeon to St. Luke's Hospital and St. Mary's Hospital for Children; Consulting Surgeon to the Home for Incurables.

1903. Mayo, Charles H., A.M., M.D., LL.D., D.Sc., F.A.C.S., Rochester, Minn. Professor of Surgery, Medical School,

ELECTED

University of Minnesota; Surgeon to Mayo Clinic (St. Mary's Hospital).

1899. MAYO, WILLIAM J., A.M., M.D., M.D. in Chir., Trinity College, Dublin, D.Sc. (Columbia, Mich., Leeds, Harvard), LL.D. (Toronto, McGill, Pennsylvania, Maryland, Pittsburgh), F.R.C.S. (Eng., Edinburgh, Ireland), F.A.C.S., Sigma Xi, Phi Beta Kappa, 701 Fourth Street, S. W., Rochester, Minnesota. Surgeon and Chief of Staff to Mayo Clinic. *Vice-President*, 1903; *President*, 1913.

†1901. MEYER, WILLY, M.D., 700 Madison Avenue, New York. Emeritus Professor of Surgery in the New York Post-Graduate Medical School; Consulting Surgeon to the Lenox Hill Hospital, Post-Graduate Hospital, the New York Infirmary for Women and Children, New York Skin and Cancer Hospital, Hospital for Deformities and Joint Diseases, Montefiore and Glens Falls Hospital.

1919. MILLER, ROBERT TALBOTT, A.B., M.D., Johns Hopkins Hospital, Baltimore, Md. Associate Professor of Surgery, Johns Hopkins University; Associate Surgeon, Johns Hopkins Hospital.

1917. MITCHELL, CHARLES F., M.D., 332 South Fifteenth Street, Philadelphia, Pa. Surgeon-in-Chief, Pennsylvania Hospital; Attending Surgeon to the Germantown and the Bryn Mawr Hospitals; Consulting Surgeon, Chestnut Hill Hospital.

1912. MITCHELL, JAMES F., A.B., M.D., 1344 Nineteenth Street, Washington, D. C. Clinical Professor of Surgery, Georgetown and George Washington Universities; Chief Surgeon, Emergency Hospital. *Vice-President*, 1920.

1924. MIXTER, CHARLES G., S.B., M.D., 270 Commonwealth Avenue, Boston. Assistant Surgeon, Children's Hospital, Boston.

†1893. MIXTER, SAMUEL JASON, S.B. (Massachusetts Institute of Technology), M.D. (Harvard), 219 Beacon Street, Boston. Consulting Surgeon to Massachusetts General Hospital and to Massachusetts Charity Eye and Ear Infirmary. *President*, 1916; *Member of Council*, 1917–19.

1920. MIXTER, WILLIAM JASON, S.B., M.D., F.A.C.S., 180 Marlborough Street, Boston, Massachusetts. Assistant Visiting Surgeon, Massachusetts General Hospital; Assistant in Surgery, Graduate School of Medicine, Harvard University; Surgeon to the Staff, Anna Jaques Hospital, Newburyport;

ELECTED

Consulting Surgeon, Leonard Morse Hospital, Natick, Charitable Eye and Ear Infirmary, Boston, Cable Memorial Hospital, Ipswich; Lieut.-Col., Med. O.R.C.

†1896. Monks, George Howard, A.B. (Harvard), M.D. (Harvard), M.R.C.S. (Eng.), 51 Commonwealth Avenue, Boston. Consulting Surgeon, Boston City Hospital; Professor of Oral Surgery, Harvard Dental School. *Vice-President*, 1909.

1914. Moschcowitz, Alexis V., M.D., 925 Madison Avenue, New York, N. Y. Professor of Clinical Surgery, College of Physicians and Surgeons, Columbia University; Attending Surgeon, Mount Sinai Hospital; Consulting Surgeon, Israel Zion Hospital.

†1904. Mudd, Harvey, G., M.D., Humboldt Building, St. Louis, Mo. Emeritus Clinical Professor of Surgery, Medical Department, Washington University; Surgeon to St. Luke's Hospital; Consulting Surgeon to the Barnard Free Skin and Cancer Hospital. *Vice-President*, 1920.

1919. Müller, George P., M.D., 1930 Spruce Street, Philadelphia, Pa. Professor of Clinical Surgery, University of Pennsylvania; Surgeon to the University and Misericordia Hospitals.

†1913. Murphy, Fred T., A.B., M.A., M.D., 2248 Penobscot Building, Detroit, Michigan.

†1898. Murray, Francis W., M.D., 7 West Forty-third Street, New York City. Consulting Surgeon, New York Hospital, St. Luke's Hospital, Paul Kimball Hospital, Lakewood, and Goshen Emergency Hospital.

1903. Neilson, Thomas R., M.D., 1937 Chestnut Street, Philadelphia, Pa. Surgeon Emeritus to the Episcopal Hospital; Consulting Surgeon to St. Christopher's Hospital for Children; Emeritus Professor of Genito-urinary Surgery in the University of Pennsylvania.

†1900. Ochsner, A. J., B.S., M.D., F.R.M.S., (Eng.), LL.D., F.A.C.S., F.R.M.S. (Ireland) (Hon.), *President*, 2106 Sedgwick Street, Chicago. Professor of Clinical Surgery, Medical Department of University of Illinois; Surgeon-in-Chief of Augustana and St. Mary's Hospitals.

†1900. Oliver, J. C., M.D., 628 Elm Street, Cincinnati, Ohio. Emeritus Professor of Surgery, Medical College, University of Cincinnati; Surgeon to the Christ Hospital.

ELECTED

1915. Olmsted, Ingersoll, M.B., 215 James Street, South, Hamilton, Ontario. Surgeon to City Hospital.

1923. Palmer, Dudley White, B.S., M.D., F.A.C.S., 707 Race Street, Pearl Market Bank Building, Cincinnati, Ohio. Associate Professor of Clinical Surgery, University of Cincinnati, Medical Department; Attending Surgeon, Cincinnati General Hospital and Christ Hospital.

†1899. Parham, Frederick William, M.D., 3513 Prytania Street, New Orleans, La. Professor of General Clinical Surgery, New Orleans Polyclinic (Post-Graduate Department of Tulane University of Louisiana); Chief of Staff to Charity Hospital and to Touro Infirmary.

†1893. Parmenter, John, M.D., Ridgecroft, Geneva, N. Y. Late Professor of Clinical Surgery, Medical Department, University of Buffalo.

1908. Peck, Charles Howard, M.D., *Treasurer*, 30 W. Fiftieth Street, New York City. Professor of Clinical Surgery, Columbia University; Senior Attending Surgeon to the Roosevelt Hospital; Consulting Surgeon to French Hospital, Memorial Hospital and Hospital for Ruptured and Crippled, New York City; Vassar Brothers Hospital, Poughkeepsie, N. Y.; White Plains Hospital, White Plains, N. Y.; Stamford Hospital, Stamford, Conn.; Greenwich Hospital, Greenwich, Conn.

1919. Percy, Nelson Mortimer, M.D., F.A.C.S., 2106 Sedgwick Street, Chicago, Ill. Professor of Clinical Surgery, Medical Department of the University of Illinois; Attending Surgeon to the Augustana and St. Mary's of Nazareth Hospitals.

1917. Phemister, Dallas B., B.S., M.D., 122 South Michigan Avenue, Chicago, Ill. Associate Professor in Surgery, Rush Medical College; Associate Attending Surgeon, Presbyterian Hospital.

†1889. Pilcher, Lewis Stephen, A.M., M.D., LL.D. (Dickinson and University of Michigan), 145 Gates Avenue, Brooklyn, N. Y. Consulting Surgeon to the St. John, Norwegian, Jewish, Bushwick and Deaconess Hospitals. *Vice-President*, 1893, 1914; *President*, 1918; *Member of Council*, 1919–21.

1914. Plummer, Samuel C., A.B., M.D., 4539 Oakenwald Avenue, Chicago, Ill. Attending Surgeon, St. Luke's Hospital; Chief Surgeon, C. R. I. & P. Railway. Co.

ELECTED

1914. Pool, Eugene Hillhouse, A.B., M.D., 107 East Sixtieth Street, New York, N. Y. Attending Surgeon, New York Hospital; Clinical Professor of Surgery, Columbia University; Consultant Surgeon to French Hospital, New York Orthopedic, Manhattan State Hospital, Home for Incurables and United Hospital at Portchester; New York Eye and Ear, and Hospital for Ruptured and Crippled.

1904. Porter, Charles Allen, A.M., M.D., 116 Beacon Street, Boston, Mass. Chief of the West Surgical Service of the Massachusetts General Hospital; Homans Professor of Surgery, Harvard Medical School.

1915. Porter, Miles Fuller, M.A., M.D., Walnut Place, Fort Wayne, Ind. Professor of Surgery in the Indiana University School of Medicine; Surgeon to St. Joseph's Hospital; Consulting Surgeon in the Indiana School for Feeble-minded Youths.

1908. Primrose, Alexander, C.B., M.B., C.M. (Edin.), M.R.C.S. (Eng.), 100 College Street, Toronto, Canada. Consulting Surgeon to the Toronto General Hospital and Consulting Surgeon to the Hospital for Sick Children, Toronto; Dean of the Faculty of Medicine and Professor of Clinical Surgery in the University of Toronto. *Vice-President*, 1922.

1923. Richardson, Edward P., A.B., M.D., Massachusetts General Hospital, Boston, Mass. Assistant Professor of Surgery, Harvard Medical School; Chief of Third Surgical Service, Massachusetts General Hospital.

1924. Ritchie, Harry P., Ph.B., M.D., 914 Lowry Building, St. Paul, Minn. Associate Professor of Surgery, University of Minnesota Medical School; Attending Surgeon, University Hospital, Minneapolis; Chief, Department of Surgery, Wilder Dispensary, St. Paul; Staff of St. Luke's and Miller Hospitals, St. Paul.

1901. Rixford, Emmet, B.S., M.D., 1795 California Street, San Francisco, Cal. Professor of Surgery in Stanford University School of Medicine; Surgeon to the San Francisco and Lane Hospitals; Member of the Society of Clinical Surgery. *Vice-President*, 1905.

‡1882. Roberts, John B., A.M., M.D., 1520 Spruce Street, Philadelphia. Emeritus Professor of Surgery in the University of Pennsylvania Graduate School of Medicine. *Vice-President*, 1888 and 1894; *Treasurer*, 1892–93; *President*, 1920.

ELECTED

1924. Rodman, J. Stewart, M.D., 1309 Medical Arts Building, Philadelphia. Professor of Surgery, Woman's Medical College of Pennsylvania; Surgeon-in-Chief, Woman's Medical College Hospital; Attending Surgeon, Bryn Mawr Hospital; Associate Surgeon, Presbyterian Hospital; Major, M.R.C.

1917. Rowan, Charles J., A.B., M.D., Iowa City, Iowa. Professor and Head of Department of Surgery, College of Medicine, State University of Iowa; Director of Surgery, University Hospital.

1922. Royster, Hubert A., A.B., M.D., Raleigh, N. C. Surgeon to Rex Hospital; Surgeon-in-Chief to St. Agnes Hospital; Consulting Surgeon to Dix Hill State Hospital for the Insane.

†1882. Rushmore, John Dikeman, M.D., 129 Montague Street, Brooklyn, N. Y. Professor of Operative and Clinical Surgery, Emeritus, Long Island College Hospital; Surgeon to St. Peter's Hospital; Consulting Surgeon to Long Island College Hospital, Kings County Hospital, Swedish and Lutheran Hospitals.

†1909. Scudder, Charles L., M.D., A.B., Ph.B., 209 Beacon Street, Boston, Mass. Consulting Surgeon, Massachusetts General Hospital; former Assistant Professor of Surgery, Harvard University Medical School.

1919. Seelig, M. G., A.B., M.D., 4952 Park View Place, St. Louis, Mo. Professor of Clinical Surgery, Washington University School of Medicine; Assistant Surgeon, Barnes Hospital; Visiting Surgeon to the Jewish Hospital.

†1902. Shepherd, Francis J., M.D., LL.D., F.R.C.S. (Eng.) F.R.C.S. (Edin.) (Hon.), F.A.C.S. (Hon.), 152 Mansfield Street, Montreal, Canada. Emeritus Professor in McGill University; Consulting Surgeon to the Montreal General and Royal Victoria Hospitals, Montreal.

1923. Shipley, Arthur M., M.D., Sc.D., 1827 Eutaw Place, Baltimore, Md. Professor of Surgery and Head of the Department of Surgery, University of Maryland; Chief Surgeon, University Hospital, City Hospitals and Sydenham Hospital; Visiting Surgeon, Union Memorial Hospital, Hospital for Women of Maryland, Mercy Hospital and Maryland General Hospital.

1920. Sistrunk, Walter E., M.D., F.A.C.S., Rochester, Minn., Surgeon to Saint Mary's, Kahler and Colonial Hospitals

ELECTED

(Mayo Clinic), Associate Professor of Surgery, Mayo Foundation, University of Minnesota.

1918. SMITH, REA, M.D., The Medical Office Building, 1136 West Sixth Street, Los Angeles, Cal. Surgeon, Hospital of the Good Samaritan, Methodist Hospital; Los Angeles General Hospital, California Lutheran Hospital.

‡1895. SOUCHON, EDMOND, M.D., 2403 St. Charles Avenue, New Orleans, La. Professor of Anatomy and of Clinical Surgery, Tulane University; Surgeon to Charity Hospital; Consulting Surgeon to the Eye, Ear, Nose and Throat Hospital. Honorary Fellow of American College of Surgeons; former President of Louisiana State Board of Health, 1898–1905. Founder of the Souchon Museum of Anatomy at Tulane University. *Vice-President*, 1899.

1919. SPEED, KELLOGG, S.B., M.D., F.A.C.S., 122 South Michigan Avenue, Chicago, Ill. Assistant Professor of Clinical Surgery, Rush Medical College of the University of Chicago; Associate Attending Surgeon, Presbyterian Hospital; Attending Surgeon, Cook County, Provident and Highland Park Hospitals.

1922. SPEESE, JOHN, M.D., 2032 Locust Street, Philadelphia, Pa. Associate Professor of Surgery, Graduate School, University of Pennsylvania; Surgeon to the Presbyterian Hospital, Children's Hospital, and the Philadelphia Home for Incurables.

1918. STARR, CLARENCE LESLIE, M.B., M.D., LL.D., 182 Bloor Street, West, Toronto, Canada. Professor of Surgery, University of Toronto; Surgeon-in-Chief, Toronto General Hospital; Consultant in Surgery, Hospital for Sick Children; Surgeon to St. John's Hospital; Lieut.-Col.-Consultant, C.A.M.C.

1914. STARR, FREDERIC NEWTON GISBORNE, C.B.E., M.B., M.D., C.M., 112 College Street, Toronto, Canada. Professor of Clinical Surgery, University of Toronto; Consulting Surgeon to Hospital for Sick Children, and Surgeon to St. John's Hospital for Women; Senior Surgeon, Toronto General Hospital.

1915. STEWART, GEORGE DAVID, M.D., 61 West Fiftieth Street, New York. President, New York Academy of Medicine; Professor of Surgery, New York University Medical School;

ELECTED

Visiting Surgeon to St. Vincent's and Bellevue Hospitals; Consulting Surgeon to Hospital for Ruptured and Crippled of New York; to St. Joseph's Hospital, Yonkers; to St. Mary's Hospital, East Orange, N. J.; to South Side Hospital, Babylon, L. I.; to Englewood Hospital, Englewood, N. J., and to the United Hospital, Portchester, N. Y.

†1909. STILLMAN, STANLEY, M.D., *Vice-President*, Stanford University Hospital, San Francisco, Cal. Professor of Surgery, Stanford University Medical School; Commander, M.C., U.S.N.R.F.; Surgeon to Lane and Stanford University Hospitals.

1923. ST. JOHN, FORDYCE BARKER, B.S., M.D., Presbyterian Hospital, 41 East Seventieth Street, New York City. Associate Professor of Surgery, College of Physicians and Surgeons; Visiting Surgeon to the Presbyterian Hospital.

1909. SUMMERS, JOHN EDWARD, M.D., Bankers Reserve Life Building, Nineteenth and Douglas Streets, Omaha, Neb. Professor of Clinical Surgery, University of Nebraska Medical School; Surgeon to the University Hospital and to the Clarkson Memorial and the Douglas County Hospitals, Omaha, Neb.

1920. TAYLOR, ALFRED S., B.P., A.M., M.D., 115 W. Fifty-fifth Street, New York. Consulting Surgeon, Fordham Hospital; Consulting Neurological Surgeon to St. Luke's Hospital, N. Y.; Neurological Surgeon, Hospital for Ruptured and Crippled; Consulting Surgeon to Memorial Hospital, New York City, Tarrytown Hospital, New York, White Plains Hospital, New York and Overlook Hospital, Summit, N. J.

†1900. TAYLOR, WILLIAM J., M.D., 1825 Pine Street, Philadelphia. Surgeon to the Orthopædic Hospital and Infirmary for Nervous Diseases; Consulting Surgeon to St. Agnes' Hospital, to the West Philadelphia Hospital for Women and to the Woman's Hospital; Member of the International Surgical Society.

1916. TERRY, WALLACE I., B.S., M.D., 380 Post Street, San Francisco, Cal. Professor of Surgery, University of California; Chief Surgeon, University of California Hospital, San Francisco.

1909. THOMPSON, JAMES E., M.B., B.S. (London), F.R.C.S. (Eng.), F.A.C.S., 3224 Broadway, Galveston, Texas. Professor of Surgery in the University of Texas; Surgeon to the John Sealy Hospital. *Vice-President*, 1922.

ELECTED

1920. Tinker, Martin B., B.S., M.D., Savings Bank Building, Ithaca, N. Y. Surgeon to the Ithaca City Hospital and the Cornell Infirmary.

1918. Truesdale, Philemon Edwards, M.D., 151 Rock Street, Fall River, Mass. Surgeon to St. Anne's and Highland Hospitals; Consulting Surgeon to Fall River City Hospital.

†1882. Vander Veer, Albert, A.M., M.D., Ph.D., LL.D., 28 Eagle Street, Albany, N. Y. Senior Consulting Surgeon, Albany Hospital; Consulting Surgeon, Albany Free Dispensary Association; Benedictine Hospital, Kingston, N. Y., and Champlain Valley Hospital, Plattsburgh, N. Y. *President*, 1905; *Vice-President*, 1898; *Member of Council*, 1907.

1910. Vander Veer, Edgar Albert, Ph.B., M.D., 28 Eagle Street, Albany, N. Y. Attending Surgeon, Albany Hospital; Consulting Surgeon, Champlain Valley Hospital and Benedictine Hospital, Kingston, N. Y.

1902. Vaughan, George Tully, M.D., LL.D., 1718 I Street, Washington, D. C. Professor of Principles and Practice of Surgery and Head of the Surgical Department, Georgetown University; Chief Surgeon, Georgetown University Hospital; Surgeon, Tuberculosis Hospital; Consulting Surgeon, Government Hospital for the Insane, Washington, D. C., U. S. Public Health Service and Washington Asylum Hospital; Commander, M. C., Reserve Force, U. S. N.

1921. Vaughan, J. Walter, A.B., M.D., 987 East Jefferson Avenue, Detroit, Mich. Associate Attending Surgeon to Harper Hospital; Surgeon, Detroit Diagnostic Hospital.

1920. Verdi, William Francis, A.M., M.D., 27 Elm Street, New Haven, Connecticut. Clinical Professor of Surgery, Yale University School of Medicine; Chief Surgeon, Hospital of St. Raphael; Attending Surgeon, New Haven Hospital; Consulting Surgeon, St. Vincent's Hospital, Bridgeport; St. Mary's Hospital, Waterbury; Middlesex Hospital, Middletown.

1922. Vincent, Beth, A.B., M.D., 295 Beacon Street, Boston, Mass. Assistant Visiting Surgeon, Massachusetts General Hospital; Consulting Surgeon, Infants Hospital, Boston.

1918. Wainwright, Jonathan M., A.B., A.M., M.D., 912 Clay Avenue, Scranton, Pa. Surgeon-in-Chief, Moses Taylor

ELECTED

Hospitals (Scranton and Buffalo); Chief Surgeon, D. L. & W. R. R. Co. and Glen Alden Coal Co.

†1891. Walker, Edward W., M.D., 214 West Seventh Street, Cincinnati, Ohio. Emeritus Professor of Clinical Surgery Miami Medical College; Surgeon, Deaconess Hospital; President of Board of Health, Cincinnati, Ohio.

1909. Walker, John B., M.D., 51 East Fiftieth Street, New York City. Consulting Surgeon to Bellevue Hospital and Hospital for Ruptured and Crippled, New York; Professor of Clinical Surgery, College of Physicians and Surgeons, Columbia University, N. Y.

†1882. Warren, John Collins, M.D., LL.D. (Jefferson, Harvard and McGill), Hon. F.R.C.S. (Eng. and Edin.), 58 Beacon Street, Boston, Mass. Moseley Professor of Surgery, Emeritus, Harvard University; Consulting Surgeon to the Massachusetts General Hospital; Member, Harvard Cancer Commission. *President*, 1896; *Vice-President*, 1890; *Member of Council*, 1897.

†1896. Watson, Francis Sedgwick, A.B., M.D., 92 Marlborough Street, Boston, Mass. Surgeon to the Boston City Hospital; Instructor in Genito-urinary Surgery, Harvard Medical School.

1913. Watts, Stephen H., M.A., M.D., LL.D., Department of Surgery, University of Virginia, Charlottesville, Va. Professor of Surgery and Gynecology in the University of Virginia; Surgeon-in-Chief, University of Virginia Hospital.

†1889. Weir, Robert F., A.M., M.D., Hon. F.R.C.S. (Eng.), Cor. Mem. Surg. Soc., Paris, 16 East Ninety-Sixth Street, New York. Professor of Surgery in the College of Physicians and Surgeons, Columbia University; Surgeon to the Roosevelt Hospital. *President*, 1899; *Member of Council*, 1893–1896; 1901–1903.

†1892. Wharton, Henry R., A.M., M.D., Berwyn, Pa. Consulting Surgeon to the Presbyterian Hospital; Emeritus Surgeon to the Children's Hospital.

1921. Whipple, Allen O., B.S., M.D., Presbyterian Hospital, 41 East Seventieth Street, New York, N. Y. Professor of Surgery, Columbia University; Director of Surgical Service, Presbyterian Hospital.

1921. Williams, Hugh, A.B., M.D., 301 Beacon Street, Boston, Mass. Assistant, Harvard Graduate School of Medicine; Visiting Surgeon, Massachusetts General Hospital.

ELECTED

†1914. Winslow, Randolph, A.B., M.D., A.M., LL.D., St. Johns College and University of Maryland, F.A.C.S., 1900 Mt. Royal Terrace, Baltimore, Md. Emeritus Professor of Surgery, University of Maryland.

1918. Wood, Alfred Conard, M.D., 2035 Walnut Street, Philadelphia, Pa. Assistant Professor of Surgery, University of Pennsylvania; Surgeon to the University, Philadelphia General, Howard and Rush Hospitals.

†1901. Woolsey, George, M.D., 117 East Thirty-sixth Street, New York City. Professor of Clinical Surgery, Cornell University Medical College; Consulting Surgeon to Bellevue Hospital, Memorial Hospital, Peekskill Hospital, Peekskill, N. Y., and St. John's Riverside Hospital, Yonkers, N. Y.

1916. Yates, J. L., Ph.B., B.S., M.D., F.A.C.S., 141 Wisconsin Street, Milwaukee, Wis. Attending Surgeon, Milwaukee County Hospital, and Columbia Hospital.

SENIOR FELLOWS OF THE ASSOCIATION

Abbe, Robert, M.D., 40 West Fifty-ninth Street, New York.

Armstrong, George E., M.D., C.M., C.M.G., LL.D., D.Sc., 320 Mountain Street, Montreal.

Barton, James M., A.M., M.D., 32 North New Hampshire Avenue, Atlantic City, N. J.

Binnie, John F., M.A., M.B., C.M., 917 Rialto Building, Kansas City, Mo.

Blake, John Babst, A.M., M.D., 311 Beacon Street, Boston, Mass.

Blake, Joseph A., A.B., PhB., M.A. (Hon.), M.D., F.A.C.S., 116 East Fifty-third Street, New York City.

Bradford, E. H., A.B., M.D., 253 Newbury Street, Boston, Mass.

Brewer, George E., M.D., LL.D., F.A.C.S., F.R.C.S. (Ireland), 19 East Sixty-fifth Street, New York City.

Bunts, Frank E., M.D., Clinic Building, Cleveland, Ohio.

Carmalt, William H., A.M., M.D., 261 St. Ronan Street, New Haven, Conn.

Carson, Norman Bruce, M.D., 303 Humboldt Building, St. Louis, Mo.

Coley, William B., M.D., The Medical Chambers, 114 East Fifty-fourth Street, New York City.

Crile, George W., M.D., Euclid Avenue and Ninety-third Street, Cleveland, Ohio.

Cushing, Hayward Warren, A.B., M.D., 70 Commonwealth Avenue, Boston, Mass.

Da Costa, J. Chalmers, M.D., 2045 Walnut Street, Philadelphia, Pa.

Dennis, Frederic Shepard, M.D., F.R.C.S. (Eng.), 62 East Fifty-fifth Street, New York, N. Y.

Elliott, John Wheelock, A.M., M.D., 124 Beacon Street, Boston, Mass.

Estes, William Lawrence, A.M., M.D., Bethlehem, Pa.
Eve, Duncan, A.M., M.D., Eve Building, Nashville, Tenn.

Finney, John M. T., M.D., 1300 Eutaw Place, Baltimore, Md.
Freeman, Leonard, B.S., M.A., M.D., 424 Metropolitan Building, Denver, Col.
Gay, George W., A.M., M.D., Chestnut Hill, Boston, Mass.

Harte, Richard H., M.D., C.M.G., 1503 Spruce Street, Philadelphia.
Huntington, Thomas W., A.B., M.D., LL.D., 960 Mills Building, San Francisco, Cal.
Hutchinson, James P. M.D., Media, Pa.

Johnson, Robert W., A.B., M.D., 101 W. Franklin Street, Baltimore, Md.
Jonas, August F., M.D., 512 McCayne Building, Omaha, Neb.

Kammerer, Frederic, M.D., Lenox Hill Hospital, New York City.
Keen, W. W., A.M., M.D., LL.D., Hon. F.R.C.S. (Eng., Edin. and Ireland), Hon. Ph.D. (Upsala), Hon. ScD. (Jefferson and Harvard), Hon. F.A.C.S., The Touraine, 1520 Spruce Street, Philadelphia, Pa.

‡Le Conte, Robert G., A.B., M.D., 2000 Spruce Street, Philadelphia, Pa.

McArthur, L. L., M.D., Ph.C.S., 122 S. Michigan Boulevard, Chicago, Ill.
Martin, Edward, A.M., M.D., LL.D., D.Sc., Media, Pa.
Mastin, Wm. M., M.D., LL.D., northeast corner Joachim and Conti Streets, Mobile, Ala.
Meyer, Willy, M.D., 700 Madison Avenue, New York City.
Mixter, Samuel Jason, S.B., M.D., 219 Beacon Street, Boston, Mass.
Monks, George H., A.B., M.D., M.R.C.S., 51 Commonwealth Avenue, Boston, Mass.
Mudd, Harvey G., M.D., Humboldt Building, St. Louis, Mo.
Murphy, Fred T., M.D., Penobscot Building, Detroit, Mich.
Murray, Francis W., M.D., 7 West Forty-third Street, New York, N. Y.

Ochsner, Albert J., B.S., F.R.M.S. (Eng.), M.D., LL.D., F.A.C.S., 710 Sedgwick Street, Chicago, Ill.
Oliver, J. C., M.D., 628 Elm Street, Cincinnati, Ohio.

PARHAM, FREDERICK WILLIAM, 3513 Prytania Street, New Orleans, La.

PARMENTER, JOHN, M.D., 519 Franklin Street, Buffalo, N. Y.

PILCHER, LEWIS S., A.M., M.D., LL.D., 145 Gates Avenue, Brooklyn, New York.

ROBERTS, JOHN B., A.M., M.D., 313 South Seventeenth Street, Philadelphia, Pa.

RUSHMORE, JOHN D., M.D., 129 Montague Street, Brooklyn, N. Y.

SCUDDER, CHARLES L., A.B., PH.B., M.D., 209 Beacon Street, Boston, Mass.

SHEPHERD, FRANCIS J., M.D., LL.D., Hon. F.R.C.S. (Edin.), 152 Mansfield Street, Montreal, Canada.

‡SOUCHON, EDMUND, M.D., 2403 Saint Charles Avenue, New Orleans, La.

STILLMAN, SANLEY, M.D., Stanford University Hospital, San Francisco, Cal.

TAYLOR, WILLIAM J., M.D., 1825 Pine Street, Philadelphia, Pa.

VANDER VEER, A., A.M., M.D., PH.D., LL.D., 28 Eagle Street, Albany, N. Y.

WALKER, EDWARD W., M.D., 214 West Seventh Street, Cincinnati, Ohio.

WARREN, J. COLLINS, M.D., LL.D., Hon. F.R.C.S. (Eng. and Edin.), 58 Beacon Street, Boston, Mass.

WATSON, FRANCIS S., A.B., M.D., 92 Marlborough Street, Boston, Mass.

WEIR, ROBERT F., A.M., M.D., Hon. F.R.C.S. (Eng.), 1155 Park Avenue, New York, N. Y.

WINSLOW, RANDOLPH, A.B., M.D., A.M., LL.D., F.A.C.S., 1900 Mt. Royal Terrace, Baltimore, Md.

WHARTON, HENRY R., A.M., M.D., Berwyn, Pa.

WOOLSEY, GEORGE, M.D., 117 East Thirty-sixth Street, New York City.

HONORARY FELLOWS OF THE ASSOCIATION

ELECTED

1907. Ballance, Sir Charles Alfred, K.C.M.G., C.B., M.V.O., M.B., M.S. (Lond.), F.R.C.S., 106 Harley Street, London, W. Consulting Surgeon to St. Thomas's Hospital and to the National Hospital for Paralysis and Epilepsy, Queen Square.

1919. Bastianelli, Raffaelle, Viale della Regina 231, Rome, Italy; Consulting Surgeon, Hospitale Rome; Professor of Surgery, Royal University, Rome and Director of the Clinic.

1919. Bowlby, Sir Anthony A., K.C.B., K.C.M.G., K.C.V.O., F.R.C.S., D.S.M., (U. S. A.), Hon. F.A.C.S., 25 Manchester Square W., London, England. Consulting Surgeon to St. Bartholomew's Hospital and to Queen Alexandra's Military Hospital.

1922. Chutro, Dr. Pedro, University of Buenos Aires, Argentina. Professor of Surgery, (Retired), University of Buenos Aires; Honorary Professor, University of Madrid, Spain; Fellow, Royal Academy of Medicine of Spain, of the Académie de Médecine de Paris, of the Surgical Association of Buenos Aires, of the Société de chirurgie de Paris; Honorary Fellow, Association of Military Surgeons of the United States.

1916. Depage, Prof. Dr. Antoine, Hon. F.R.C.S. (Eng.), 75 Avenue Louise, Brussels, Belgium.

1919. Hartmann, Dr. Henri, Hon. F.R.C.S. (Eng.), 4 Place Malesherbes, xvii^{e} Paris, France. Professeur de Clinique Chirurgicale, Chirurgien de l'Hôtel Dieu. Membre de L'Académie de Médicine.

1920. Keen, W. W., A.M., M.D., LL.D., Hon. F.R.C.S. (Eng., Edin. and Ireland), Hon. Ph.D. (Upsala), Hon. Sc.D. (Jeff. and Harv.), Hon. F.A.C.S., 1729 Chestnut Street, Philadelphia.

1918. Keogh, Sir Alfred, Hy., G.C.B., Hon. F.R.C.S., The Bungalow, East Grinstead, England.

ELECTED

1922. Leriche, Dr. René, 13 Quai Claude Bernard, Lyons, France.

1918. Makins, Sir George, G.C.M.G., C.B., LL.D. (Cambridge and Aberdeen), F.R.C.S.

1920. Manoury, Gabriel, Dr., 26 Avenue de Maréchal-Manoury, Chartres, France, Chirurgeon de l'Hôtel-Dieu de Chartres; Associé Nationale de l'Académie de Médecine.

1903. Mayo-Robson, Col. Sir Arthur, K.B.E., C.B., C.V.O., F.R.C.S., D.Sc., Broadoak, Seale, Surrey, Eng.; Consulting Surgeon, King Edward VII Memorial Hospital, Windsor, and Leeds Infirmary. Emeritus Professor of Surgery, University of Leeds; late Vice-President, Royal College of Surgeons and Hunterian Professor; Consulting Surgeon, M.E.F. (Dardanelles, Egypt, etc.), twice mentioned in dispatches; afterward Consulting Surgeon, Southern Council; member of Consulting Council, War Office; Consulting Surgeon, Croix Rouge Française; Chevalier Legion d'Honneur; Knight of of the Order of St. John of Jerusalem.

1908. Moynihan, Sir, B. G. A., Bart., K.C.M.G., C.B., M.S. (Lond.), F.R.C.S., 33 Park Square, Leeds, England. Professor of Surgery, University of Leeds; Senior Surgeon, Leeds General Infirmary.

1907. Myles, Sir Thomas, M.D., F.R.C.S.I., 33 Merrion Square N., Dublin, Ireland.

1912. Stiles, Sir Harold Jalland, K.B.E., LL.D., D.Sc., M.B., F.R.C.S. (Edin.), F.A.C.S., F.R.C.S.I., 9 Great Stuart Street, Edinburgh, Scotland. Regius Professor of Clinical Surgery, University of Edinburgh; President of the Royal College of Surgeons of Edinburgh; Surgeon to the Edinburgh Royal Infirmary and Consulting Surgeon to Chalmers Hospital and to the Royal Edinburgh Hospital for Sick Children, Edinburgh.

1924. Taylor, Sir William, Dublin, Ireland.

1918. Tuffier, Th., M.D., Hon. F.R.C.S. (Eng.), 42 Avenue Gabriel, Paris, France. Professeur agrégé de chirurgie; Membre de l'Académie de Médecine; Président de la section thérapeutique du Cancer, Hôpital cité du Midi, 48 boulevard de Clichy.

ELECTED

1920. Wallace, Sir Cuthbert, K.C.M.G., C.B., D.S.M. (U. S. A.), M.B., B.S. (London), F.R.C.S., (Eng.), 26 Upper Wimpole Street, W., London, Eng.; Surgeon to St. Thomas's Hospital, London; Dean of the Medical School.

1920. Walther, Charles, M.D., 68 Rue de Belle-Chasse, Paris, France. Professeur Agrégé à la Faculté de Médecine, Chirurgien Honoraire des Hôpitaux; Membre de l'Académie de Médecine.

DECEASED FELLOWS OF THE ASSOCIATION

Name	Date of death
Agnew, D. Hayes	March 22, 1892
Allen, Dudley Peter	January 6, 1915
Allis, Oscar Huntington	May 16, 1921
Annandale, Thomas	1907
Ashhurst, John, Jr.	July 7, 1900
*Atlee, John Light	October 1, 1885
Beckman, Emil Hessel	November 7, 1916
Benham, Silas Nelson	November 3, 1890
*von Bergmann, Ernst	Died since 1906 Meeting
*Bigelow, Henry Jacob	October 30, 1890
*Billings, John Shaw	March 11, 1913
*Billroth, Albert Christian Theodor	January 5, 1894
Bontecou, R. B.	1906–1907
Bosher, Lewis C.	September 12, 1920
Briggs, William Thompson	June 13, 1894
Brinton, John Hill	March 18, 1908
Bristow, Algernon Thomas	March 26, 1913
Brock, Hugh W.	April 24, 1882
Bryant, Joseph Decatur	April 7, 1914
*Bryant, Thomas	1914–1915
Bull, William Tillinghast	February 22, 1909
Burrell, Herbert Leslie	1910
Byrd, William Andrew	August 14, 1887
Cabot, Arthur Tracy	November 4, 1912
Campbell, Henry Fraser	December 15, 1891
Cheever, David Williams	December 27, 1915
*Chiene, John	May 29, 1923
Cole, Charles Knox	February 27, 1920
Conner, Phineas Sanborn	March 26, 1909
Cunningham, Francis Deane	September 11, 1885
*Czerny, Prof. Dr. Vincenz	October 3, 1916
Dandridge, Nathaniel P.	1911–1912
Davis, Gwilym G.	June 16, 1918

* Honorary.

Name	Date of death
Davis, John Staige	July 18, 1885
Dawson, William Wirt	February 16, 1893
Dugas, Louis Alexander	October 19, 1884
Dunn, James Henry	June 16, 1904
Dunott, Thomas Justus	May 20, 1893
*Durham, Arthur Edward	May 7, 1895
*Erichsen, Sir John Eric	September 23, 1896
*von Esmarch, Friedrich	1907
Fenger, Christian	March 7, 1902
Ferguson, Alexander Hugh	October 20, 1911
Forbes, William Smith	December 17, 1905
Fowler, George Ryerson	February 6, 1906
*Friedrich, Paul Leopold	January 5, 1916
Gaston, James McFadden	November 17, 1903
Gerrish, Frederic Henry	September 8, 1920
Gerster, Arpad G.	March 11, 1923
*Gorgas, William C.	July 4, 1920
Greene, W. Warren	September 10, 1881
Gross, Samuel David	May 6, 1884
Gross, Samuel Weissel	April 16, 1889
Gunn, Moses	November 4, 1887
*Gussenbauer, Karl	June 19, 1903
Guthrie, George Washington	June 1, 1915
Halsted, William Stewart	September 7, 1922
Harrington, Francis B.	1914–1915
*Harrison, Reginald	1907
Hearn, W. Joseph	December 20, 1917
Hodge, H. Lenox	June 16, 1881
Hodgen, John T.	April 28, 1882
Homans, John	February 7, 1903
*Horsley, Sir Victor Alexander, C. B.	July 16, 1916
Horwitz, Orville	January 28, 1913
*Horwitz, Phineas J.	September 28, 1904
Hughes, J. C.	August 10, 1881
*Humphrey, Sir George Murray	September 24, 1896
*Hunt, William	January 5, 1897

* Honorary.

Name	Date of death
Jacobson, Nathan	September 16, 1913
Johnson, Alexander B.	September 4, 1917
Johnston, George Ben	December 20, 1916
Johnston, Christopher	October 11, 1891
King, Alfred	June 4, 1916
Kinloch, Robert Alexander	December 23, 1891
*Kocher, Theodor	July 27, 1917
Lane, Levi Cooper	February 18, 1902
*von Langenbeck, Bernhardt	September 30, 1887
Levis, Richard J.	November 12, 1890
*Lister (Lord), Joseph	February 11, 1912
Little, James Lawrence	April 4, 1885
Lutz, Frank Joseph	March 24, 1916
McCann, James	1893
McCosh, Andrew J.	December 3, 1908
McGraw, Theodore A.	September 6, 1921
McGuire, Hunter	September 19, 1900
McMurtry, Lewis Samuel	February 1, 1924
McRea, Floyd W.	August 13, 1921
*MacCormac, Sir William	December 4, 1901
MacDonald, Willis Goss	December 30, 1910
*Macewen, Sir William	March 22, 1924
Mackenzie, Kenneth Alexander James	March 15, 1920
MacMonagle, Beverly	May 22, 1912
Markoe, Francis Hartman	1907
Marks, Solon	1916
Mastin, Claudius Henry	October 3, 1898
Mears, J. Ewing	May 28, 1919
Michael, J. Edwin	December 7, 1895
*Mikulicz-Radecki, John V.	June 17, 1905
Miles, Albert B.	August 5, 1894
Millard, Perry H.	February 1, 1897
Miller, Truman W.	May 31, 1900
*Moore, Edward Mott	March 3, 1902
Moore, James E.	November 2, 1918
Mudd, Henry Hodgen	November 20, 1899
Mumford, James Gregory	October 18, 1914
Munro, John C.	December 6, 1910
Murphy, John B.	August 11, 1916

* Honorary.

Name	Date of death
de Nancrède, C. B. G.	April 12, 1921
Nichols, Edward Hall	July 6, 1922
Norris, Basil E.	October, 1895
*von Nussbaum, J. N.	October 31, 1890
*Ollier, Leopold	1900–1901
Oviatt, Charles W.	October 31, 1912
Owens, John E.	December 21, 1922
*Paget, Sir James	December 30, 1899
Pancoast, William H.	January 5, 1897
Park, Roswell	February 15, 1914
*Parker, Willard	April 25, 1884
Parkes, Charles T.	March 28, 1891
Parkhill, Clayton	January 16, 1902
*Péan, Jules E.	January 30, 1898
Peck, Washington F.	December 30, 1891
Pilcher, Paul Monroe	January 4, 1917
Porter, Charles Burnham	May 21, 1909
Porter, William Gibbs	January 30, 1906
Powers, Charles A.	December 23, 1922
*Pozzi, Samuel	1918
Prewitt, Theodore F.	October 17, 1904
Prince, David	December 19, 1889
Ransohoff, Joseph	March 10, 1921
Reed, Thomas Baird	April 1, 1891
Richardson, Maurice Howe	July 31, 1912
Richardson, Tobias Gibson	May 26, 1892
Rodman, William Louis	March 8, 1915
Ross, George Gorgas	December 27, 1922
Sabine, Thomas Taunton	August 23, 1888
Sayre, Lewis A.	1900–1901
*Schede, Max	1902
Senn, Nicholas	January 2, 1908
Sherman, Harry M.	1921.
*Sims, J. Marion	November 13, 1883
*Smith, Stephen	August 26, 1922
Squire, Truman Hoffman	November 27, 1889
Stewart, Francis T.	February 4, 1920

* Honorary.

Name	Date of death
*Terrier, Louis Felix	1908
*Thiersch, Karl	April 28, 1895
Thompson, Joseph Ford	February 1, 1917
Thompson, Joseph W.	March 30, 1886
*Thomson, Alexis	March 4, 1924
Thomson, William	August 3, 1907
Tiffany, Louis McLane	October 23, 1916
Varick, Theodore Romeyn	November 23, 1887
*Verneuil, Aristide A. S.	June 11, 1895
*von Volkmann, Richard	November 28, 1889
Watson, Beriah Andrew	December 22, 1892
Weeks, Stephen Holmes	September 1, 1909
Weist, Jacob Rowland	May 14, 1900
*Wells, Sir Thomas Spencer	January 31, 1897
White, James William	April 24, 1916
Wight, Jarvis S.	November 16, 1901
Willard, DeForest	October 14, 1910
Wood, James R.	May 4, 1882
*Yandell, David Wendell	May 2, 1898

* Honorary.

PRESENTATION OF A WREATH IN MEMORY OF PASTEUR

THE following address was made in Paris, July 12, 1923, by Dr. C. L. Gibson, on the occasion of the presentation of a wreath in memory of Pasteur. This presentation was made by Dr. Gibson in accordance with a resolution of the Council of the American Surgical Association. The presentation was acknowledged by Roux, head of the Pasteur Institute. Others present included Calmette, Tuffier and Manoury, the latter two Honorary Fellows of the Association. With the wreath there was a large silk ribbon, on which was inscribed "Homage of the American Surgical Association." This ribbon is preserved in the collection of memorabilia.

MONSIEUR ROUX; MESSIEURS LES MEMBRES ÉTRANGERS DE L'AMERICAN SURGICAL ASSOCIATION:

Je remplis avec émotion la mission d'honorer la mémoire du grand Français, que l'American Surgical Association m'a confiée.

Nous reconnaissous profondément que l'existence d'une association de Chirurgie comme la nôtre est possible seulement par les travaux de ce grand chercheur de la vérité scientifique.

Tout ce que je pourrais dire n'ajouterait rien à la réputation de Pasteur le savant. Mais je me réjouis beaucoup, en fervent de la France et des Français de saluer la mémoire de Pasteur, le grand patriote le représentant surtout de ces belles qualités qui ont toujours été, seront toujours, la grande force de la France.

C'est pour nous un exemple frappant que le plus grand bienfait que le monde ait jamais reçu fût fourni par un homme d'origine soi-disante modeste, et ne sortant pas d'une lignée de travailleurs intellectuels. Que ses débuts furent pénibles et qu'à un moment donné il semblait destiné à retourner plutôt à la vie dure et simple de ses ancètres. Mais ces qualités de travail patient, l'ardeur d'accomplir, héritage de ces travailleurs de la terre le poussèrent toujours vers son but.

Il faut aussi se souvenir des difficultés matérielles qui s'opposèrent à ses travaux, difficultés qui décourageraient tout être non doué de foi scientifique. Aussi faut-il admirer cette bravoure qui a surmonté les limitations du corps, et défié par de longues années productives une maladie ordinairement signalant la fin d'une carrière.

C'est alors à l'homme, digne représentant de la belle et vieille souche française, autant qu'au plus grand bienfaiteur du monde que je porte le tribut de l'affection et l'estime des travailleurs de la jeune nation américaine.

REPORT OF COMMITTEE ON ARCHIVES

DR. CHARLES A. POWERS*

I SHOULD like to revive an old custom, perhaps fallen a little into disuse, and say a few words in memory of a deceased fellow, Charles Andrew Powers, a former President, and for ten years wise and active on the Council. No Fellow ever gave more of his time and thought to the Association than he; and I stand here to mourn with you the loss of a friend and review a few of the personal qualities that endeared him to us all.

Dr. Powers started practice under brilliant auspices, for he was chosen by Dr. William T. Bull, then reaching the pinnacle of his fame, as assistant and companion in his work. Had he continued in this field undoubtedly he would have become one of the greatest of metropolitan surgeons, but the great white plague touched him with her cold hand and he was sent to the Adirondacks. He improved there and again essayed work in New York. This was followed by a more serious breakdown and he was ordered to live in Colorado. There, when he recovered his strength a little, he started life over again; made light of his exile, achieved the distinction of being the best-known surgeon in the Rocky Mountain district and gathered about him an ever-increasing circle of devoted friends. His footsteps trod no royal road, and his character was formed, softened and humanized by his indomitable courage in overcoming all obstacles presented. With him it was a constant fight to keep alive and in condition to work. He was exiled from his home and the companions of his youth. He lost one of man's greatest heritages, free will to go where one pleases. He was disappointed in married life and the establishment of a home life. In all the avenues we choose for a happy existence a stone wall confronted him. Adversity seemed ever dogging at his heels. Yet he rose superior to all these trials and tribulations, emerging in midlife a man of great courage, profound in wisdom and judg-

* Prepared by Dr. Robert G. Le Conte, and read by Dr. Richard H. Harte at the meeting in Rochester, Minn., 1923.

ment, loyal to a cause or a friend beyond the exigencies of justice, gentle, filled with human kindness, but fearless in exposing an untruth. He was a man who was accorded universal respect and affection from those who knew him. When the Lusitania was sunk, his feeling of loyalty to the cause of humanity was so strong he volunteered his services and served in the American Hospital at Neuilly. When the United States entered the War he was enrolled in the Medical Department of the Army. He did his work well; but health again failed him and he was invalided home a month before the Armistice. His services were recognized by the army with the Distinguished Service Medal, and by France and Belgium with orders of merit. When the war was over he was too feeble to resume again the arduous duties of a surgical life: but did his labors cease? Oh, no; for he accepted the trying duties of President of the National Society for the Control of Cancer; and, like all his work for humanity, he left it better for his endeavor in its behalf. He died in harness on the eve of Christmas, suddenly, as he was sending greetings and messages of cheer to his friends. He occupies a grave with our national heroes in Arlington, buried with full military honors and the emblem of meritorius service on his breast from the country he loved so well.

To epitomise his life from early manhood. It started on a bed of roses, with fragrant opening buds, each one of which in turn withered, leaving the naked thorns to torment him; but, like the staff of Tannhäuser, they sprouted again, not as a sign of redemption, but to display to us the flower of courage, of loyalty and of friendship.

No words of mine can do adequate justice to the loss we have sustained; and, mornful though we are, we can rejoice in what he has left behind for us to profit by and in the manner of his death. May the memory of our friend remain enshrined in our hearts and his noble example be an inspiration in our lives, that we also may have said to us, "Well done, thou good and faithful servant."

R. G. Le C.

DR. LEWIS SAMUEL McMURTRY

Dr. Lewis Samuel McMurtry died of pneumonia at his home in Louisville, February 1, 1924. He had been a member of the American Surgical Association since 1903. He was a pioneer in the new abdominal surgery, inaugurated in the 90's, and exemplified those traits of leadership which made him an outstanding figure in surgery and its development in this country. The traditions of his city, state,

and the part the South has contributed to American surgery were worthily sustained in his active life as a teacher, a writer and an organizer. In rare measure Dr. McMurtry typified gentility and good-fellowship. He was a gifted and convincing speaker, which was enhanced by a most charming personality.

Dr. McMurtry was born at Harrodsburg, Ky., September 14, 1850, and lived continuously in his native state. His family was among the early settlers of Kentucky and came of a Scotch ancestry. He spent his childhood in Lancaster, Garrard County, Kentucky, and was educated at Center College, Danville, receiving his A.B. degree at nineteen years of ago and his A.M. three years later. He was graduated in medicine from the medical department of Tulane University, of New Orleans, and spent a year following his graduation as intern in the Charity Hospital of Louisiana.

He entered the practice of medicine at Danville. In 1881 he was elected to the chair of anatomy in the Kentucky School of Medicine at Louisville. In 1883 and 1884 he pursued special studies in pathologic histology and clinical surgery in New York and Philadelphia, and in 1889 he spent several months in Europe with Mr. Lawson Tait.

That year he removed his residence permanently to Louisville and devoted himself exclusively to the practice of gynecology and abdominal surgery. He was professor of gynecology and abdominal surgery in the Hospital College of Medicine from 1890 until the Louisville schools merged into the University of Louisville in 1908. During that period he was president of the Faculty of the Hospital College of Medicine, and after the merger occurred he was president of the University of Louisville until two years ago, when he was elected president of the State Board of Health of Kentucky. He was continually on the staff of the Louisville City Hospital, St. Mary and Elizabeth's Hospital and the Gray Street Infirmary and Norton Infirmary. In 1895 he acquired the Jenny Cassiday Hospital for Women, in Louisville and conducted it as a private hospital until 1906. In 1908 he was elected president of the staff of the Norton Infirmary, and served in that capacity until two years ago, when he voluntarily retired.

He was elected president of the American Medical Association, at Atlantic City, in 1904, and presided at the Portland meeting. He was a member of the first Board of Trustees, rendering valuable service in founding and establishing the *Journal*.

He was president of the Kentucky State Medical Society in 1889, and was chairman of the committee which erected the monument to Dr. Ephraim McDowell, at Danville, which was unveiled by Dr. Samuel D. Gross.

Dr. McMurtry was one of the most active members of the Southern Surgical Association, and was elected its president in 1891. In 1893 he was made president of the American Association of Obstetricians and Gynecologists.

He was a frequent contributor to current medical literature and contributed a chapter on "Surgery of the Uterus" in the *International Text-book of Surgery*, and made numerous contributions to Reed's *Text-book of Gynecology*.

He was an honorary member of the Philadelphia Obstetrical Society and Fellow of the Edinburgh Obstetrical Society, Fellow of the British Gynecology Society, and one of the founders of the International Periodical Congress of Gynecology and Obstetrics. He was an honorary member of the Medical Society of the State of New York and a founder of the Pan-American Medical Congress, acting as president of the Section on Obstetrics and Gynecology at the first meeting of the latter, at Washington, in 1893.

He performed the first recorded successful operation for acute intestinal obstruction and gall stones ever done in Louisville. It could be properly stated that he ushered the newer era in surgery into his community.

In 1914 he visited the clinics of Europe and Great Britain, with the American Gynecological Club, and delivered the dedicatory address at the unveiling of the statue of George Washington in Budapest.

He was a founder and one of the Board of Governors of the American College of Surgeons.

During the World War he organized the first hospital unit which reached the front from Louisville, under the command of Major Irvin Lindenberger. He was military aid to the Governor of Kentucky during the period of our participation in hostilities.

He was a speaker at the centennial celebration of Pasteur's anniversary, held at the Bellevue-Stratford Hotel, Philadelphia, December 31, 1922. His address on that occasion, "Pasteur's Relation to Surgery," has since been translated and preserved in the archives at the Pasteur Institute in Paris.

In February, 1923, he delivered the Hodgen Lecture, entitled "The Birth of Modern Surgery," before the St. Louis Medical Society St. Louis, Mo. W. D. H.

SIR WILLIAM MACEWEN

With the death of Sir William Macewen, professor of surgery in the University of Glasgow, British surgery has lost one of its most

brilliant exponents. He died on March 22, 1924, at his residence in Glasgow, from pneumonia following influenza. He was seventy-six years of age, and within a fortnight of his death was actively engaged in the discharge of his University and other duties.

It is impossible to compress into a few lines even a summary of his character and work. He was one of the most arresting figures which has appeared on the stage of British medicine since John Hunter and Lister. And he drew his deepest inspiration from these men of outstanding genius. Hunter led him into the paths of the naturalist for aid in the solution of some of his problems. It was in the changes in the antler of the deer that Macewen found the strongest proof of his theory of the regeneration of bone.

Lister was his teacher in surgery, and his influence on him was immediate and definite. It formed the pivot on which his success revolved. Macewen saw clearly the value to surgery of its association with scientific inquiry, and he therefore proceeded to lay a solid foundation in a wide knowledge of anatomy and pathology. With great acumen he realized, at an early stage of its study, that Listerism was only a phase and that asepticism was the true principle upon which to act.

In 1877, at the early age of twenty-nine years, he was appointed visiting surgeon to the Royal Infirmary, Glasgow, and it was during the fifteen years he was there that he achieved some of his greatest triumphs. First among these was his mastery over sepsis in surgical wounds which he obtained in large measure through his courage and vision. He realized some years before his contemporaries did that the infection of wounds might take place through other avenues than the air around the wound, so he set about reorganizing his department, and this was carried out in a manner characteristic of him. He swept aside the old order of things, and insisted upon his own individual authority as supreme in his own department. The instruments supplied by the hospital were discarded and his own private equipment substituted, and the instruments, now his own property, were guarded from contamination by the curious with scrupulous care. The behavior of the catgut ligature he determined from numerous tests on dogs.

Although a man of few words and a strict disciplinarian he nevertheless evoked from his assistants and nurses unstinted loyalty and coöperation. As in the case of all great reformers, he had to meet opposition, and conflicts were frequent between him and the hospital directors who were wont to lean on authority and custom. But he was resolute and had faith like another great master of surgery who also opposed authority, Ambroise Paré.

The measure of his success in the application of aseptic principles to the treatment of operation wounds may be gathered from the table given in his work on osteotomy for genu valgum, published in 1880. 835 osteotomies were performed with only 3 deaths—1 from acute pneumonia, 1 from tuberculous meningitis and the third from diphtheria. Few surgeons today, with the aid of gloves and sterilizers, could surpass these results in respect of wound healing and a low mortality. In his operation for the cure of inguinal hernia, 1886, he gives further proof of his confidence to prevent the entrance of germs into the wounds at operations. He separated the sac and transposed it to the area of the internal ring. In no instance did the sac slough nor did suppuration take place. Another proof was given in a demonstration in the Royal Infirmary, Glasgow, in 1886, before a number of surgeons attending his clinic, some of whom had come from afar, when he showed the twenty-second consecutive case of a major amputation in which healing had been secured with one dressing.

Macewen was now in a position to enter the field of experimental and scientific surgery, and to apply to the human subject the benefits of the researches of the anatomists and physiologists, and to add to the knowledge they had gained. With what success he accomplished all this the surgical world knows, and it has acclaimed his merits. His name is linked with the great advances in surgery in the past fifty years; but of his contributions, those on the regeneration of bone and on the surgery of the brain and spinal cord are among his best.

By his colleagues and many hundreds of graduates he will be remembered as a great surgeon and as one who devoted his great gifts to the search after truth. R. H. P.

JOHN CHIENE, C.B., M.D., LL.D., D.Sc., F.R.C.S.E.

To one who has been pupil, house surgeon and subsequently, for ten years, a university assistant to the late Emeritus Professor Chiene, it is a privilege, as well as a labor of love, to endeavor to pay tribute to his memory. That he possessed such a strong and many-sided personality, that he was such a well-known and widely esteemed citizen, so highly reputed as a surgeon, so famous and beloved as a teacher, as well as so full of humanity, makes the task no easy one.

Chiene was appointed to the chair of systematic surgery in 1882. His reputation as an extramural teacher had been so great that the writer purposely delayed taking out a course of lectures in surgery until Chiene had become professor. One cause of his great success

as a lecturer was the fact that he never wrote out his lectures in full. They were always thought out afresh each year. I well remember, when his university assistant, the mosaic appearance of his printed syllabus, which was crossed in all directions with jottings he had made at various times. Although he took a keen interest in the science and progress of surgery, he was not a laborious reader of the subject. He had however, a great gift for weeding out the grain from the chaff of surgical literature. Like his great masters, Syme and Lister, he was an individualist; his lectures were founded almost entirely on his own observations and experience. He presented his facts in a simple, original and logical manner, and with such a fine sense of proportion that the student got a mental perspective of surgery and a grip of its principles well calculated to prove of lasting value to him in his future.

As a clinical teacher, Chiene was equally successful and popular. It was a treat to see with what consummate skill he used to brush aside details and demonstrate how the cases illustrated the surgical principles he so forcibly laid down in his systematic lectures. No one could have been more earnest and conscientious in clinical teaching, and so convinced was he of its value that it was a serious offence for a student to appear in his wards without his note-book and pencil ready to hand.

Chiene could not be regarded as a rapid or brilliant operator, nor would he have wished to be so considered. Having been a demonstrator, first under Goodsir and then under Turner, he was a good anatomist and could operate rapidly when the necessity arose; but he always put safety first, and anything in the shape of playing to the gallery was repulsive to him. His success as an operating surgeon was due in no small measure to the judgment—he knew what to do as well as how to do it. Unfortunately for him, however, in spite of his strong character, and in spite of the fact that he had no lack of surgical courage, he was possessed of a sensitive and, in some respects, a nervous temperament, which made the strain of his operative work all the heavier; moreover, the anesthetic was always a source of anxiety to him. It was this trait in his character that made him happier in the lecture room and wards than in the operating theater.

When Lister left Edinburgh it was Chiene who bore the brunt of the battle of "Listerism," which was being waged in the Edinburgh Royal Infirmary, just as it was Hector Cameron who fought it in Glasgow. Chiene's victory was all the more creditable, considering the strength of the forces arrayed against him. Chiene was brought up in the school of anatomical surgery which prevailed at the time. His intimate association with Lister and the discovery of the tubercle

bacillus by Koch in the same year as he was appointed professor of systematic surgery, combined with his keen foresight and wide outlook, were doubtless the main influences which determined him to set up a laboratory of bacteriology and surgical pathology in the university. This was the first teaching bacteriological laboratory in the United Kingdom, and by establishing it, Chiene set the example, in the academic teaching of surgery, of cultivating the subject as a science, so that its art might be better taught and promoted. Although he was not himself a laboratory worker, he spared neither time nor money to encourage research by his assistants.

During the latter part of his occupancy of his chair, Chiene began to feel the effects of his strenuous life, and though destined yet to live beyond the allotted span, his honest and conscientious nature prompted him to sheathe his sword and hand over the command to a surgeon of the younger generation. That he felt the wrench keenly and sorrowfully is only human and it was a great disappointment to his many friends that he so seldom emerged from the retirement of his charming retreat overlooking the Brunsfield golf-links and the banks of the Forth.

Doctors, as a rule, have little time or inclination for public work outside their professional duties, but of Chiene it may truthfully be said that, in spite of his arduous routine work, he gave freely of his time and thought on behalf of many a project concerned with the welfare of the community. His commanding presence, his strong yet genial countenance and his popularity made him a familiar figure to the citizens of Edinburgh. Chiene was more than a surgeon and teacher—he was a man of broad and statesman-like vision; he "reflected the human spirit always on the nobler side." He possessed "those finer qualities of head and heart which count for so much in life" and are such a valuable asset, especially to a clinical teacher. It is safe to assert that his great natural ability, his strong individuality and lovable personality would have assured him success in any sphere of life. He was an apostle of common sense, was able to leaven his lectures and clinics with an amount of shrewd and helpful everyday philosophy much appreciated by his pupils. The methods which he introduced and the influence which he exercised on the Edinburgh Medical School have been far-reaching in their beneficence. His name will go down to posterity as a man of wide sympathies and high ideals, as well as a great teacher and sound surgeon.

Chiene was a famous football player and the first president of the Scottish Football Union. To the present generation he was a familiar figure on the golf course and on the curling pond. Like many of my contemporaries, I owe some of my happiest days and reminiscences

to the foursomes in which I was privileged to take part with my old and revered teacher in the days of Old Luffness and later at Machriehanish. The way Chiene played "with his head," the moral support he gave his partner and the miracles he performed with his "little black spoon" made him a tower of strength to his partner. He knew his Bible as well as most ministers, and many a text was quoted in his classroom and wards in support of a principle or rule of conduct. His favorite text for the golf links was, "In quietness and confidence shall be your strength"—the best motto also for a surgeon. If his partner should miss a short putt, he would say, "Never mind Harold, just think how happy you have made Harry or Francis" (both of whom, alas, have crossed the bar before him). He loved to play a good, old, foursome, because, he said, it teaches us to "bear one another's burdens." He was a very "clubbable" man and used to advise the young practitioners to join a social club as well as the local medical society. Full of pawky Scottish humor and a good reconteur, he was always a great asset to the several medical dining clubs for which Edinburgh has long been famous. Although not an orator in the true sense of the word, he was an original and attractive speaker.

Professor Chiene was particularly happy in his home life, which was very dear to him. Deep sympathy will be felt for his family and for his devoted and courageous wife, who so ably and efficiently shared with him the burdens as well as the joys of a life so strenuous and useful, so full of honor, service and achievement. H. J. S.

HENRY ALEXIS THOMSON, C.M.G., M.D.

Alexis Thomson and the writer were fellow-students and graduates of the same year; we were house surgeons together in the Edinburgh Royal Infirmary; we embarked on the same life-work; we took almost parallel routes, both of which were equally steep; we had the same ambitions and much the same objective. During the race he was always a hurdle in front of me, and had he retained his health he would have outstripped all his rivals, and there would have been almost no limit to his achievements. All through our lifetime of healthy rivalry not a single cloud ever collected to shut out even temporarily the brightness which illuminated our friendship, which was of the most intimate nature and which will ever remain a priceless memory of my whole professional career. Small wonder, therefore, that I am grateful for the privilege which has been accorded me

by paying tribute to his memory, difficult as it is to do justice to such a brilliant and complex personality.

Alexis Thomson was an Edinburgh man born and bred, and I was fortunate as a student in being admitted to his home circle. He was one of seven sons, and his mother, who was highly cultured, a good linguist and a great reader, knew full well the value of education, and did not hesitate to make the necessary sacrifices to enable her family to benefit from it. Young Alexis was accordingly sent for a year to Germany and a year to Switzerland to complete his preliminary education. As a student he was universally popular, and the esteem in which he was held is evidenced by the fact that he was elected one of the presidents of the Royal Medical Society. Rowing was his chief outdoor hobby, an exercise for which his splendid physique was well suited.

After graduating (in 1885) we both became house surgeons—Thomson to the late Mr. John Duncan and I to the late Professor Chiene. Thomson—or Alexis, as he was always called—was the life and soul of the mess and the ringleader in many revelries. Even then he showed evidences of that brilliant wit and repartee for which he subsequently became so famous and which made his lectures so attractive.

On leaving the Royal Infirmary he became resident physician to the Royal Edinburgh Hospital for Sick Children, which in those days was next door to the infirmary. After our resident appointments we were anxious to remain in Edinburgh, and both of us decided to go in for surgery. The bread-and-butter problem, however, faced us both in an acute form. Thomson became a junior assistant in the pathological department under the late Professor Greenfield and I a demonstrator of anatomy under the late Sir William Turner. In those days junior assistantships in the University did not mean a living wage—far from it. To eke out a bare existence, therefore, we went into partnership in conducting a coaching class for the second professional examination. Thanks to Alexis's popularity and gifts as a teacher, we established a record class, at any rate in point of numbers.

It was shortly after this that Thomson did his splendid work on tuberculous affections of the bones and joints, and it was then that he derived so much benefit from his mother's foresight in sending him to school on the Continent. I well remember the delight and keenness with which he read the voluminous work of König (then professor of surgery in Göttingen) on this subject. The infirmary provided him with a rich supply of material for his investigations, with the result that he was able to add to König's clinical observa-

tions a more thorough knowledge of the pathology and morbid histology of these conditions. His researches were embodied in a thesis for which he was awarded a gold medal.

Early in his career he became assistant surgeon to the Royal Edinburgh Hospital for Sick Children, and while he was assistant surgeon to the Edinburgh Royal Infirmary he was also surgeon to the Deaconess Hospital. He was fortunate, therefore, in gaining experience in operative work at a comparatively early age. During this time he had the advantage of working in succession in the wards and operating theaters of Mr. John Duncan, Professor Annandale and Professor Chiene. His great ability and tireless industry enabled him to take full advantage of his opportunities. From John Duncan he acquired the habit of painstaking clinical investigation, logical reasoning and accurate induction. His association with Professor Annandale as his assistant surgeon gave him a unique opportunity of witnessing and assisting a man who was universally recognized as one of the most dextrous and resourceful as well as one of the most daring operators of the early antiseptic era. His subsequent assistant surgeonship with Professor Chiene, whom he succeeded, gave him the opportunity of tempering boldness in operating with prudence and of acquiring from him the art of utilizing clinical material to illustrate surgical principles. Chiene, along with Sir Halliday Groom, was famed for his systematic teaching. Thomson deserves to be classed with them. Although his style of lecturing was very different, it was equally effective. He presented his subject in admirable perspective and with great lucidity; his diction was at once precise, concise, penetrating and illuminating; his ripe and varied experience, his profound knowledge of his subject and of human nature, combined with his ready wit and wisdom, made his lectures unusually attractive. His sympathies were always on the side of youth. He loved to draw his breath in a student atmosphere, whether in or outside the classroom. He took a personal interest in every member of his class, knew their capabilities, had a smile for their foibles and a sigh for their difficulties. He had great experience as an examiner, and woe betide the man who tried to hoodwink him. He had a genius for making friends, especially among the younger generation. The students' esteem and love for him amounted to hero-worship. They spoke of him as "our Alexis," and it is safe to say that it will be a long time, if ever, ere a successor be found to fill his place in the hearts of the students.

During the World War, when he served as consulting surgeon to the Third Army he fell a victim to trench fever, and was never the same man again. Fortunately, his intellect remained as clear as

ever, and he continued to take the same interest in his teaching, but his old friends saw less and less of him, and when he was struck down and unable to continue the work he loved and did so well, it was not to be wondered that he lost heart. It was hoped that a trip to Algeciras would be beneficial, but, alas, this was not to be, and the end came suddenly. His eminence as a surgeon was so outstanding and so universally recognized that it would be superfluous for me to dwell on it. Always a student, a good linguist and a great reader, he had an exceptionally wide knowledge of the literature of surgery. His own contributions, which were original and of great value, served to enlarge the boundaries of our knowledge of scientific surgery in several directions. Of the *Manual of Surgery*, of which he was joint author, it is no disparagement to others to say that as a text-book for students and practitioners there is no better in any language.

Alexis Thomson had a keen appreciation of the joys of play as well as of work. He was an ideal holiday companion. Among the happiest days of my life are those I shared with him in the residency of the Royal Infirmary, on the mountain peaks of the Isle of Arran, on the golf courses of East Lothian, especially Old Luffness, and on the continents of Europe and America, both alone and with our many mutual American friends, especially with the members of the Society of Clinical Surgery and with the Fellows of the American Surgical Association.

H. J. S.

ROYAL SOCIETY OF MEDICINE
11 JUN
1925

ADDRESS OF THE PRESIDENT

A BIPOLAR THEORY OF THE NATURE OF CANCER

By GEORGE W. CRILE, M.D.
CLEVELAND, OHIO

It is a high privilege to stand as the successor of the long line of distinguished surgeons who have held the office of President of this, the oldest surgical society in America. This list and the lists of the past and present members of this society include the names of many who have made notable contributions to the advancement of surgery. A review of their accomplishments has made it no easy matter to decide upon the subject for the address which custom requires.

The history of medicine has shown that the field of cancer has been a hospitable cemetery for the hopes of the sponsors of the many theories, and the problem presented by the grimmest enemy of all is still a mystery. It is not necessary to enumerate before this group any statistics to remind us of our obligation to prosecute the study of cancer by every method of investigation at our command. For these reasons, therefore, I have decided to offer a discussion of a theory as to the nature of cancer which is suggested by a new line of investigation. It is not expected that this theory will escape the common lot of cancer theories, but whatever happens to the theory certain facts will probably survive.

Cancer originates and lives only in the living—not in the non-living. It follows that our problem is bound up in the

struggle for survival between the cancer cell and the normal cell, and the logical point of attack, therefore, is the nature and structure of the normal cell as compared with the nature and structure of the cancer cell. It is proposed, on this occasion, to search the normal and cancer cells for such physical differences as may explain the superior ability of the cancer cell to multiply at the expense of other tissues in which it grows.

The best-known example of growth energy is that initiated by fertilization in reproduction. The outstanding facts regarding fertilization which may throw light on the cancer problem are:

1. The spermatozoön has the properties of the nucleus of the ovum with which it unites.

2. The spermatozoön may be said to reinforce the nucleus, and, as a consequence,

3. The quiescent negative ovum flares up in active metabolism and growth, and in consequence shows a striking change in its internal structure—and it assumes electrical properties, *i. e.*, electricity is a constant phenomenon from the moment of fertilization so long as the life of the new individual lasts.

This comparison of the processes of the multiplication of cancer cells with that of fertilized cells is no new conception, the similarity of the nuclear changes having even led to the supposition by some that malignant processes actually were the result of some form of fertilization. Moreover, the cyclic variations in the growth of tumors correspond to the cyclic changes in nuclear and mitotic activities which have been observed in protozoöns.

The whole histologic picture of malignancy indicates that it is primarily nuclear in origin, as is suggested especially by the large nuclear-plasma ratio which is maintained either by the size of a single nucleus or multiple nuclei; by nuclear hyperchromatism in the active stages and by the shrinkage of the nuclei in the degenerating or necrosed areas.

On the basis that the processes of cell division in cancer are analogous to the processes of cell division in fertilized cells, we shall report certain biophysical researches and point out certain pertinent facts tending to show that cancer falls within the

domain of the electrochemical or bipolar theory, which we believe applies to normal living processes.

Certain analogies between cancer and the pyogenic infections may aid in this interpretation. Cancer cells multiply, bacteria multiply, and each finds restraint in certain tissue. Neither cancer nor the pyogenic infections commonly attack tissues of high oxidative capacity; thus neither cancer nor pyogenic infections primarily attack the heart muscle, the voluntary muscles, the cortex of the brain, the normal thyroid gland, the liver, the parenchyma of the kidney, the spleen, etc. No enzyme, no specific chemical property, has been found to account for this fact. These are tissues of high chemical activities; these organs are homogeneous in structure and their unit cells are closely approximated and bathed in fluid—in other words, these organs are concentrated cell suspensions. Neither infection nor cancer attack successfully the anatomically and physiologically intact surface layers of cells like the skin and mucous membranes, the latter in turn being an electrically charged cell-suspension system; they attack rather the less cellular structures which normally are protected by cellular layers.

Our first generalization, then, is that cancer originates not in the *midst* of a cell suspension, such as the cellular organs, but at the boundary points between highly cellular and less cellular structures. These less cellular structures—subcutaneous, submucous—are successfully attacked by cancer or infection only when the cellular defense is broken down. In the case of a pyogenic invasion a single break in the line of defense may be sufficient for entrance; cancer depends rather upon the gradual lessening of the defense which results from the frequent breaking down and building up. Once the rapid infection or the slower cancer has passed this first line of defense, each follows the path of least resistance, namely, the lymphatic channels and the connective tissue, rather than attacking the solid cellular organs.

Another analogy between cancer and infection is found in the fact that each obeys laws of cell division and growth which apparently are the same as those which govern the cells of the host.

Both cancer and infection are repulsed by vigorous metabolic activity within the defending structures; thus, as we have already noted, the heart muscle, the voluntary muscles and the normal thyroid are relatively immune. To this fact we may add the significant fact that bacteria do not attack the most active part of the cell itself; that is, the nucleus of the cell is immune to pyogenic invasion. To this statement it should be added that the cell nucleus and bacteria show a similar stain reaction. Finally, unlike the normal cells of animals, cancer cells and bacteria have no specific function—they possess only growth energy.

A consideration of these facts suggests the following biophysical interpretation based upon the argument which was presented in the Murphy oration as published in the current issue of *Surgery, Gynecology and Obstetrics*. The conclusions drawn from that argument are: (1) That the difference between the living and the non-living depends upon the accumulation of free energy on the dielectric lipoid films which surround the nucleus and the cytoplasm and the numerous spherules within the cells; (2) that the charges on these films are derived from oxidation; (3) that oxidation within the cells is governed by the difference in energy potential between the nucleus and the cytoplasm; and (4) that, therefore, both the growth and the special function of cells are dependent on their structure and their energy potential. Thus the area of oxidating surface in the nucleus of a cell as compared with the area of oxidating surface in the cytoplasm is another way of expressing the nuclear-plasma relationship, and signifies that the larger the nucleus in comparison with the cytoplasm the greater the energy potential of the cell. Thus, if two cells have an identical organization, an identical energy potential, then, with respect to each other in the competition for nutrition, their chances are even; but if in one of two adjacent cells the size and organization of the nucleus is such as to give it a greater capacity for oxidation, hence a greater demand for nutrition, then the cell of comparatively low oxidative capacity will suffer in the competition and will break down in starvation.

As we have stated above, in cancer cells the nucleus plasma

relation resembles that of fertilized cells. Before fertilization the ovum in itself is so lacking in organization, and hence in oxidative capacity, that there is apparently little or no difference in potential between its nucleus and its cytoplasm—it carries no electric charge: it is inactive, negative. But when the nucleus of the ovum is reinforced by the nucleus-like spermatozoön there is at once established a difference in energy potential whin the cell, oxidation becomes rapid, nutrition is demanded, the size of the nucleus increases, mitosis is inaugurated, cell division occurs.

As we have stated, our interpretation of cancer assumes that the difference between the cancer cell and the neighboring cells of lower potential is analogous to the difference between the unfertilized and the fertilized ovum. The analogy ends, however, once the mechanism of cell division has been established, for cancer cells have little or no differentiation.

If the foregoing biophysical interpretation be correct, then cancer tissue must meet the following biophysical requirements: (1) the cancer cells must have a high capacity for the storage of electric charges and (2) the conductivity of cancer tissue must show specific variations from the conductivity of normal tissues. That is, if our assumption is correct then the lipoid films of the cancer cells and of the normal cells and the fertilized cells would take electric charges in a direct ratio to the combined surface area of their lipoid films. For instance, though in its external appearance a fertilized fish egg is apparently the same as an unfertilized egg, one would expect the former to show a higher capacity than the latter; one would expect that the capacity of cancer cells would be higher than that of normal cells; one would expect that radiation would lower the capacity of cells; one would expect to find a higher capacity in such cellular tissues as the brain, liver, muscles, adrenals, thyroid, spleen and pancreas than in such indifferent tissues as connective tissue and fat.

Our first researches along biophysical lines were a series of conductivity measurements of normal and of pathological tissues made in collaboration with Helen Hosmer, B.S., and Amy

Rowland, M.A. The clinical tissues measured included malignant and benign tumors of the breast and of the uterus, ulcer and carcinoma of the stomach, carcinoma of the rectum, malignant and benign tumors of the mouth, jaws and neck, *x*-ray burns and various types of goiters—hyperplasia, fetal adenoma, multiple adenoma, toxic adenoma, exophthalmic goiter, simple colloid goiter and thyroiditis. The following were the significant findings:

1. In all instances in which comparative measurements were made the conductivity of the malignant growth was higher than that of a normal portion of the same organ.

2. The outer growing parts of cancers showed a high conductivity in contrast with the conductivity of the central non-growing parts.

3. Among the goiters studied the highest conductivities were found in the degenerating adenomata and the malignant thyroids; the conductivities of the hyperplastic thyroids were lower and the conductivities of the colloid goiters were the lowest of any of the pathological tissues studied.

These measurements were made with an alternating current of 1000 cycles. This comparatively low frequency of current would probably find the path of lowest resistance, in large part, undoubtedly through the intercellular tissues.

Extending this line of inquiry, the theoretical requirement that cancer tissue must have a high capacity for the storage of electric charges was given to Hugo Fricke, Ph.D., and Sterne Morse, M.D., of the Department of Biophysics of the Cleveland Clinic Foundation, for investigation. The versatile mathematical mind of Dr. Fricke has derived a formula and devised an apparatus whereby frequencies ranging from 100 to 1,000,000 or more cycles can be applied to the cells under investigation. Up to the present time currents varying from 3000 to 125,000 cycles have been used. The physical estimations of the capacity of normal tissue and of benign and malignant tumors thus far made are as follows:

Up to the present time 80 tissues from 58 cases have been

investigated, including 15 carcinomata of various types, 3 sarcomata, 3 benign tumors and 16 goiters of various types. Normal tissues have ranged from 0.47 for fatty tissue and 1 to 3 for connective tissue to 10 for the normal uterus. All of the carcinomata have had a relatively high capacity ranging in the arbitrary units used from 11 to 32 in the actively growing portions of the growth. The degenerated portions of the growth have had a lower capacity and radiated tissues have been much lower, the tissue in one radiated case showing as low a capacity as 3. Thus far in every case studied the tissue in which the cancer had developed had a lower capacity than the cancer itself. This difference has been particularly marked in the case of carcinomata of the breast in which the capacity of the adjacent glandular connective or fatty tissue has often been less than one-tenth that of the malignant tissue. Among the goiters, colloid goiters have shown the highest capacity of any tissues studied—as much as 76 in one case, the average being in the neighborhood of 40. This finding is of prime significance, in view of the fact that cancer of the thyroid never develops in a colloid goiter. Adenomata and hyperplastic thyroids have, as a rule, had a low capacity for glandular tissue, which in general seemed to show a somewhat higher capacity than other tissue. Connective tissue has usually a very low value, between 1 and 3, and the capacity of fatty tissue may be as low as 0.47, while an active inflammatory process may show a capacity of 20. These measurements run parallel relatively to the conductivity measurements previously made by Miss Hosmer and Miss Rowland.

The findings in these researches suggested at once that the whole story of cancer may ultimately be derived from conductivity and capacity measurements. These findings, moreover, are in accord with the histologic picture presented by the microscope. The microscope indicates the general structure, which in turn *indicates* the capacity of the cell for work, multiplication, function, etc. A further striking parallel between the cytologic picture and biophysical findings is found in the fact that Ewing and Wood have shown that cells which have

been subjected to lethal *x*-ray or radium radiation show loss of differential stainability, and in our laboratory Morse has shown that heavily radiated tissue almost wholly loses its capacity.

Armed with these physical facts, let us see to what extent some of the well-known facts regarding cancer may be harmonized. First of all, on the basis of electric potential, implying, as it does, oxidative capacity, if two cells are side by side competing for food the one having the higher potential, such as the fertilized cell or the cancer cell, starves out, and if the higher potential—higher oxidative capacity—persists long enough, destroys the ordinary tissue. Among cells with equal capacity, such as those within the cancer itself or the daughter cells of the fertilized ovum, division occurs evenly—no one starves another. This fact explains why cancer does not arise either primarily or secondarily in that fiery furnace, the heart muscle, or in other muscles, or in the cortex of the brain, or in the thyroid gland, etc. It also indicates why, when from some cause the capacity of an epithelial cell resting on subcutaneous or submucous cells of low capacity has been increased until it is equal to the capacity resulting from fertilization, that cell will easily rob the neighboring, inactive tissues of their nutrition and will supplant them, just as the vigorous growing weed overgrows and supplants the highly differentiated, less vigorous domestic crops.

A consideration of the conditions under which cancer develops in the thyroid gland is illuminating. First, cancer almost never develops in the normal thyroid or in colloid goiters, but over 90 per cent of cancers of the thyroid arise in the fetal adenomata. Now, as has been stated above, the capacity measurements made by Morse show that both colloid goiter and the normal gland have a higher capacity than cancer of the thyroid, while the capacity of fetal adenoma is lower than that of cancer. Now, on the basis of our premise, one would have predicted that that would be the case, even though he knew nothing about the actual incidence of cancer in the thyroid gland.

Again, let us consider one of the most common sites of cancer origin—the breast. Here is an organ whose structure contains

epithelial cells, the capacity of which is low. It follows that when some circumstances bring into contact with these low-capacity epithelial cells, cells with a relatively high potential, the latter multiply at the expense of the other breast tissue. The capacity of cancer of the breast is from two to ten times higher than the capacity of normal breast tissue. The capacity of the tissue near the cancer mass is somewhat higher than that of normal tissue. In general, benign tumors have a higher capacity than that of the organ in which they grow, *e. g.*, the capacity of a fibroid is higher than that of the normal uterus.

There is a general analogy to this conception of the law governing incidence of cancer in the various tissues, in the tables of Voit, which show that in starvation the weight of the brain and of the heart muscle does not change, the reason being that these tissues, the metabolism of which is at a higher rate than that of other tissues, consume the nutrition at the expense of the others. For the same reason a fetus thrives up to the point of starvation of the mother.

This conception explains the higher incidence of cancer in old age when the generally falling metabolism would diminish the already low defense of the tissues of low capacity and lead to an inequality in an already wavering balance between the capacities of neighboring cells. Moreover, the older and the feebler the subject the slower the growth of cancer and the better the prognosis; and, *per contra*, the younger and more vigorous the subject the shorter the course and the more fatal the cancer. But youth has fewer cancers than old age. Our theory interprets this antithesis as follows: In the general activity of all tissues in youth it would be unusual to find the potential of any one cell raised above that of its equally vigorous neighbors; but once so phenomenal a cell has been produced, its growth energy would be enormous, rapid and fatal. In old age, on the other hand, with its universal decline in activity, although a cell may more readily become endowed with the equivalent of fertilization so that its potential and its capacity may become higher than those of its feeble neighbors, yet it would have a very moderate growth

energy. In fact, cancer in the aged and feeble inevitably would appear just above the low level of low vitality and in youth just above the high level of general vitality. In youth the cancer must be virile; in age it must be feebler. Thus, in experimental studies cancers are not transplanted to the muscles, nor to the liver, nor to the heart, nor to the brain, but to the more negative tissues; it is the subcutaneous quiescent breast tissue that is generally selected as the site for the graft.

If one could plant a self-limited bacterium in the nucleus of a cell, its added oxidation might augment the nucleus in a manner analogous to the augmentation of the nucleus of the ovum by the spermatozoön, so that, in consequence, cell division would be forced. Or if one could draw the nucleus out of one cell and insert it into a sister cell, thus reinforcing its nucleus, the energy potential of the latter cell would be increased, its nutrition intake increased and cell division would follow, *i. e.*, a cancer would be produced.

The interpretation of another fact is made possible by the biochemical theory, namely, the like action of *x*-ray and radium on cancer and on fertilization. The effect of radiation is to interfere with the mechanism in the cell for the creation and storage of electrical charges, an interference which as effectively prevents growth and function as does the permanent discharge of any battery.

An interpretation of still another group of facts is suggested by the biophysical theory, namely, that sarcomata appear early in life and are disseminated while epithelial growths appear later in life. A possible though admittedly not a clear interpretation of this differentiation would be that cells distributed by the blood lodge in the more vigorous oxidizing organs, such as the muscles, glands, etc., while the cells spread by the lymphatics reach the lymph glands, whose metabolism is relatively low. Therefore, the older and the feebler the patient, the more probable it is that the cancer cells, which are most likely to metastasize, will be those that reach the lymphatic glands whose flame is burning low. Again, when the potential of a cell in the high-

tension thyroid rises above that of its fellows, and in consequence multiplies at their expense in a fetal adenoma, one would expect a wide extension of these cells, and such is the case.

Certain every-day facts about treatment are also open to a biophysical interpretation. Thus, if a cancer is entirely removed early no return is seen, whereas if these electrochemical mechanisms are stimulated by injury, by partial operation, by inflammation, by chemical agents, by *x*-ray, by radium, by heat, by electricity, the resultant struggle and survival kill off the weaker cells, leaving the stronger. When a massive treatment is given any cells which survive will be the fittest, hence the return growth will be at the pace of the strongest, the fittest cancer cells, not of the less strong cells that did not survive. The combination of diminishing vigor on the one hand and a stepping up on the other, theoretically, would bring about the unbalance required for cancer.

Pyogenic Infection. We have already mentioned certain analogies between cancer and pyogenic infections. Certain further biologic principles governing infection, which appear to be the same as those governing cancer, may be cited. The resemblance between cancer and infection has been noted by many observers. Pyogenic bacteria may be regarded as free nuclei, like the fragmented nuclei seen in many unicellular organisms. If we regard the law of universal bipolarism as a necessary condition by means of which a difference in potential is created and oxidation controlled, with resultant electric charges and maintenance of potential, we interpret bacteria as free nuclei, depending for their common negative pole on the common colloids, such as mud, soil, sea-water, etc., or the colloids in the tissues and fluids of animals. Bacteria, then, will multiply as free nuclei.

A cancer cell is a bipolar mechanism within which the nucleus is the positive and the cytoplasm the negative pole; bacteria are positive poles with lymph and tissue juices as a common negative pole. According to this conception, the cancer cell and the bacterium are in a common class of high potential

invaders. Now, the bacterium like the cancer cell, must depend on its ability to compete with the cells of the organism for nutrition. It is probably a consequence of this fact that bacteria, like cancer, cannot primarily compete with the cells of the organs which have a high metabolism. Bacteria, like cancer, attack best the negative tissues, the subcutaneous tendons, the fascia, the bone and surfaces that have been irritated. Bacteria stain like nuclei; bacteria almost never attack nuclei of cells, almost never muscles, most seldom of all the heart muscle. Bacteria tend to spread by the adynamic lymphatic system rather than by the dynamic blood stream. However, as in cancer, if bacteria are potent enough, *i. e.*, have the required potential to multiply in the blood-stream, those bacteria are most apt to kill, and to kill early. We find, then, that both bacteria and cancer cells multiply at the expense of their host; both may form tumors; both cause reactions; both interfere with function; both are selective as to the attacked organs, as to invasion. Though they have much in common, they are, nevertheless, entirely different.

As to the problems suggested by this discussion, it would seem that in the capacity estimations we may have one more criterion for the diagnosis of cancer. Should this hope be realized the estimation of capacity will best be made on fresh, unstained tissue almost instantly after its removal or, when conditions permit, *in situ*. Even if this should not become a specific method of operating-room diagnosis, it will quite surely, even in its present status, supplement the microscope. We may perhaps find in this new biophysical method not only a means of diagnosis, but one of prognosis as well—low-capacity relation of the growth to the adjacent tissues would theoretically mean low malignancy, and *vice versa*. There is one more and a unique possibility, namely, that after the effects of radiation on capacity have been studied further, it may be that the capacity of a tumor may at last furnish the key to the amount of radiation required as a lethal dose; or, indeed, whether any dose that the normal tissue of the organism could endure would cure, so that futile efforts could be avoided. There are still other interesting lines of

investigation, but a discussion of these is reserved for a further report by Dr. Fricke and Dr. Morse.

Conclusion. In conclusion, one of the greatest obligations science owes to humanity is the control of cancer, and it is hoped that the suggested theory, on the one hand, and, on the other hand the new facts which have been and are being demonstrated by the more exact sciences of mathematics and physics may contribute a new point of attack. The theory has at least this advantage—that it has been formulated and developed on the basis of exact mathematical formulæ and physical laws, and in consequence can the more readily be disproved if not true, and if true, can be the more easily defended.

HOW THE TRADITION OF BRITISH SURGERY CAME TO AMERICA

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LONDON

As I am credited with knowing something of the development of surgery in Great Britain, I thought it might not be out of place if I tried to show you today how the British surgical tradition arrived in this country, a tradition which has enabled you in a very short time to build up a science of surgery which it took us many centuries to obtain. You have, indeed, learned something from us at the beginning, and our tradition was formerly of use to you. But for many years past you have developed surgery along lines laid down by yourselves to our advantage, for we have learned much by watching your progress.

Go back as far as we will in English medicine, we have always had well-defined grades of medical practitioners—the physician, the surgeon, the family doctor, the apothecary, the dentist and the quack, who usually posed as a specialist and was itinerant. I show you pictures of each of them, and you will see that they are as clearly differentiated by their dress as they were by their education and uses. No grade trenched upon the preserves of another. *The physician* was generally an ecclesiastic, with a greater knowledge of books than of practice; he was essentially a teacher of the theory of medicine. *The surgeon* was a practical man—stout-hearted, for he was willing to run risks; not always of unblemished reputation in early days and rather greedy in the matter of fees. *The family doctor*—we should call him a herbalist nowadays, for he treated his patients for the most part with infusions of plants growing in his own garden or in the

hedges; but he used his common-sense and called in a surgeon when in difficulties just as he still does. *The apothecary* was a tradesman, who made and sold the more complicated remedies, like theriac, which contained several hundred ingredients, and the plasters which it was beneath the dignity of the physicians to manufacture and was too long and expensive a job for the surgeon or family doctor to undertake. The dentist was itinerant; he traveled from place to place, carrying the teeth he had extracted in a bandolier, that all might see his skill. He announced his advent with the musical cry of "touch and go, touch and go, says kind heart the dentist," which was heard in London well on into the eighteenth century. *The quacks* were the couchers for cataract, the curers of rupture, the cutters for stone, and the bonesetters; they were a pestilent brood, with some rough skill and with a terrible mortality.

There were advantages and disadvantages in practising medicine in the early days of which I am now speaking: Our predecessors made no bad debts; they were rarely accused of malpractice, and they were, if I may dare to say so, a remarkably close trades union; they had, on the other hand, no social position, and their lot sometimes fell in very evil places.

The physician as an ecclesiastic was protected by the Church, and was usually attached to the court or to the person of some great nobleman. He was, therefore, always sure of protection. The surgeon—at any rate in London—was a member of the fellowship of surgeons, a fraternity whose members coöpted each other, and who usually selected their own apprentices. The barbers, or barber surgeons, who were the family doctors in all the large English towns, could not practise at all until they were free of the guild or company of barbers, to which they must have been apprenticed for five or seven years. The apothecaries were all members of the very important guild of pepperers or grocers, and their wares were officially inspected from time to time, and were cast into the open street if they were not up to a rather high standard of purity.

I said a little while ago that the surgeons were of necessity

stout-hearted people, though they were not always of unblemished reputation. They were stout-hearted, because if an operation were not so successful as had been represented beforehand to the patient and his friends the surgeon sometimes came to an untimely end. King John of Bohemia, for instance, from whom Edward the Black Prince is said to have taken his cognizance of three ostrich feathers at the battle of Creçy, was operated upon for cataract. The king said that he could see no better after the operation than before, so the surgeon was tied up in a sack and was thrown into the Oder. We know that similar sequelæ sometimes happened in England. It is no matter of surprise, therefore, that the surgeon was always careful to take his fee beforehand, and, as it was the custom for him to live in the patient's house until the cure was completed, it happened from time to time that he rode off some fine morning without saying where he was going when he had dressed the wound and found that it was not looking as well as he would have wished.

A somewhat lurid light is thrown upon the habits of surgeons when it is said: "A wise surgeon will do well to refrain from stealing anything while he is actually attending a patient, and he should be careful not to employ notoriously bad characters as his assistants, for these things may spoil a good operation and thus detract from the dignity of medicine. A surgeon too, should not talk to the women of the house with closed doors, whether she be mistress or servant; he should never speak improperly to her nor make eyes at her, especially in the presence of the patient. Such actions may cause a patient to lose confidence in his surgeon, and thus the operation may prove unsuccessful, because the patient has lost the good opinion he had of the operator." On the other hand, it is said, "The rich have a nasty habit of coming to the surgeon in old clothes, or, if they are properly dressed, as befits their station, they invent all kinds of excuses for beating down his fees. They say charity is a flower and think that a surgeon ought to help the unfortunate, but they never consider that a like rule is binding upon themselves. I often say to such folk," the writer adds, "well, then, pay me

for yourself and for three paupers and I will cure them as well as you. But they never make any answer."

I have only been able to find one action for malpractice in these early days, and that was in the year 1424, when a citizen of London, called William Forest, sued John Harwe, an "enfranchised surgeon," John Dalton and Simon Rolf, "barbers admitted solely to the practise of surgery," for an injury to the muscles of his right thumb. It appeared from the evidence that the plaintiff had cut his thumb and that there had been recurrent hemorrhage on six occasions, which the two barbers, practising surgery, had stopped successfully, but temporarily. It recurred a seventh time with such violence that John Harwe, the surgeon, was called in, who finally applied the actual cautery and arrested it. Harwe had fortunately told the patient in the presence of witnesses that the thumb would never be as useful as it had been, and the man had said that he would rather suffer mutilation of his hand than bleed to death. The arbitrators, therefore, had no hesitation in giving their award in favor of the defendants, which was to "impose perpetual silence on the said William about this affair; moreover, we find that the surgeon and the barbers are so free from the fault attributed to them, and have been defamed so maliciously and undeservedly that, as far as in us lies, we restore to them their good name unsullied. We further declare that any defect of the aforesaid hand thus wounded, or the mutilation, or its ugly shape, is the result of the injury occurring when the moon was dark and in a bloody sign, namely, under the very malevolent constellation Aquarius, on the last day of last January, or else from some peculiar defect or injury in the said Forest." It must have been a comfortable time for a surgeon when a bad result could be attributed to the influence of an evil planet! We still recall this state of thought when an event is spoken of as being "ill-starred."

The family doctors went their way through the centuries without much change, but it was quite otherwise with the surgeons. They gradually came to be so completely subordinate to the physicians that they could order nothing but the com-

monest remedies unless the prescription was countersigned by the physician, and they could not perform a major operation except in the presence of a physician. The surgeon, therefore, had no knowledge at all of what would now be called inner medicine or physic, and his calling was looked upon as so much inferior that it was almost impossible for one who had been practising surgery ever to become a physician.

An attempt was made during the reign of King Henry VIII, about 1540, to raise the standard of surgery from a mere trade into a recognized profession, partly by improving the education of the apprentices and obliging them to pass examinations before they were admitted to become masters, and partly by eliminating the quacks, who flourished in great numbers. Surgeons in London, Maidstone and Nottingham took part in the struggle, but it proved futile, and when these pioneers died no one was found to fill their places, so that for another hundred years surgery remained in a subordinate position.

The régime began when the surgeon became a gentleman—that is to say, when he became educated to the level of the better classes among his contemporaries. Such men at first were few and far between. Richard Wiseman was perhaps the first, a surgeon who had seen much service in the Civil War during the middle of the seventeenth century. He died of hemoptysis, and says that he was often obliged to stop operating on account of a sudden burst of bleeding from his lungs. The illness was bad for him, but good for us, because during the periods of enforced idleness he wrote his *Several Surgical Treatises*. He tells in these treatises of the cases he had seen and, collating like with like, he draws conclusions as to their nature. His predecessors had contented themselves with the publication of individual cases of rare conditions without any useful commentary. But Wiseman was as one born out of full time, for after his death, in 1676, there was again a barren period in British surgery until the profession as distinct from the trade of surgery came into permanent existence. It began, I think, about 1711, with William Cheselden, surgeon to St. Thomas's Hospital, a skilful

lithotomist and oculist, anatomist and architect, a friend of the great literary characters of his day. The poet Pope wrote of him:

> "Late as it is I put myself to school
> And feel some comfort not to be a fool.
> Weak though I am of limb and short of sight,
> Far from a lynx and not a giant quite,
> I'll do, what Mead and Cheselden advise,
> To help these limbs and to preserve these eyes.
> Not to go back is somewhat to advance,
> And men must walk at least before they dance."

Cheselden was followed by Sharpe and Percivall Pott, surgeons and educated men, to whom John Hunter owed something. They were great operating surgeons and teachers, but they were men of their time. Hunter differed entirely. He was a great thinker, and for the first time he raised surgery in England to the dignity of a science. Where his immediate predecessors had been little more than anthropotomists and clinical surgeons, he was a comparative anatomist, a physiologist and a pioneer in surgical pathology. He was original in thought and put his ideas to the test of experiment. He was a great teacher, as well by example as by precept, and he founded a school in which you and we are, I hope, apt pupils.

You may perhaps ask why I have told you all this which at first sight has nothing to do with American surgery. It is because I wanted to show you something of our surgical tradition and how we were helped as well as hindered by its existence. Our physicians remained physicians, our surgeons remained surgeons, our family doctors remained family doctors. It has been different with you, for a large and new country, with a scattered population, had to evolve new methods and develop on different lines. Still we speak a similar language, and, although as a nation you are less homogeneous than we are, we appear to think very much along the same lines. But if we are alike in our ideas the course of development has been very different. Self-reliance has been your most marked character-

istic. Every doctor had to do everything, and for many years there was no opportunity for specializing even in the larger towns, except that, as must always happen, one man would show a bent for inner medicine, another for surgery and yet another would be more interested in midwifery. With us self-reliance was not fostered. For many hundred years every surgeon and every family doctor was obliged, under penalty of fine, imprisonment or being debarred from further practice, to "present" any patient under his care who was in danger of death or permanent disability. The presentation was made to the master and wardens of the Company of Barber Surgeons or Surgeons within three days of the serious illness or accident of any patient they were called upon to treat, and these officials were bound to attend without taking any fee during their year of office. The regulation worked well, for there is no instance of a master or his wardens having abused their position, though many family doctors and surgeons were punished for neglecting to call them in. It made, indeed, for good fellowship, and it shows incidentally how small a town even London must have been when patients who were seriously ill could be visited individually by the busy men at the head of their profession, who had also to attend to their own work. With you it would have been impossible, for the distances and the difficulties of locomotion would have prevented it, and therefore, as I have said, your conditions made for entire self-reliance. How splendidly you took it and what risks you ran! Think of Ephraim McDowell operating upon Mrs. Crawford in 1809, in a village; or Wright Post and Valentine Mott tying repeatedly the femoral, subclavian and common carotid arteries under the most primitive conditions; or of William Gibson ligaturing the common iliac in circumstances which would have appalled the surgeon of today. They had, of course, to deal with healthy bodies and the absence of those pathogenic organisms which are so much more numerous in crowded tenements than in small towns and villages. In this respect, and at this time, they were more favorably placed than our surgeons, when it was openly advertised in London that.

"Here you can get drunk for a penny, dead drunk for twopence and straw to lie upon for nothing."

The bulk of your profession at this time was poor, for money, as with us in country places, was scarce, and patients paid rather in kind than in cash. The examinations, even if they were taken, were not very searching, and your surgeons were more learned in experience than book lore, for books were so extraordinarily scarce that we read of only one or two copies of a text-book existing in a whole state. Distances were great, and a man must either ride or walk where the roads or tracks were not available for wheeled traffic, as was mostly the case. Climatic conditions were severe, and it often happened that a man was so tired at the end of his day's work—which, indeed, more often than not extended into the next day—that he was unable to make any contribution to the literature of his craft. But in spite of this a profession gradually grew up and a tradition was imported. A father who had been somewhat more successful in practice than his fellows determined that if his son was so enamoured of surgery that he *would* follow it, he should at least have a better chance than himself. He would send him to London or Edinburgh or Paris sometimes for the whole of his training, sometimes for a postgraduate course after attendance at one of the American universities. There also came to you from Europe a few anatomists and surgeons who, for a variety of reasons, chose to settle among you, and brought with them surgical knowledge from France, Switzerland, Great Britain and Ireland.

Among the earliest of these English teachers was Abraham Chovet, whose history has been so excellently elucidated by Dr. William Snow Miller. Chovet was born in 1704, and must have been a good teacher of anatomy, for he published a syllabus of his lectures in 1732 and was a demonstrator of anatomy at the Barber Surgeons' Hall in London from 1734–36, a post which was of first-rate importance in the teaching world of the time. He was thus contemporary with William Cheselden, and was antecedent to Percivall Pott, and, of course, to John Hunter. For some reason he left London rather suddenly and went first to

Barbadoes and afterward to Jamaica, whence he is said to have been driven out by a threatened insurrection of slaves. He fled to Philadelphia and began to lecture there in 1774. The lectures were delivered, he says, "on his elegant anatomical wax figures and real preparations of the parts beautifully embalmed and prepared." We may be sure that as an old man of seventy he was *laudator temporis acti*, and that what the barber surgeons had done in London in the early part of the century was good enough for him; he must have been the incarnation of the English tradition. He lived on in Philadelphia until 1790 and was the only one of the twelve senior founders of the College of Physicians who was not born in or near Philadelphia. His anatomical models appear to have been preserved until they were accidentally destroyed by fire in 1888. Stories are told of his sarcastic wit, as, for instance: "The doctor happening to be overtaken at the house of a member of the Society of Friends by a heavy shower of rain, his patient offered to lend him an overcoat, adding that he must not swear while he wore it—a habit to which he was notoriously addicted. On returning the coat the Quaker asked him, "Friend, didst thou swear while thou hadst on my coat?" "No," replied the doctor, "but there was a damnable disposition to lie."

In like manner came Granville Sharp Pattison many years later. He had a brother in Pennsylvania, and after lecturing on anatomy and physiology at Glasgow, he arrived at Philadelphia in 1819 and succeeded Dr. Physick as professor of anatomy in the University. He taught for a few years and then returned to London, to take up a similar appointment at University College. Of a wandering disposition, he came back to the United States and was appointed professor of anatomy at the Jefferson Medical College in 1832. Nine years afterward he had migrated to New York, and there he died in 1852.

There were doubtless other Englishmen who came over as teachers and brought the English methods of teaching with them, but these are two outstanding examples.

Much more numerous were the sons of successful doctors

and business men who were sent to Europe to receive or complete their medical education, and of these there is a long and very distinguished list. Here are some of their names: Wright Post, Philip Syng Physick, John Syng Dorsey, John Collins Warren, Valentine Mott, Wiliam Gibson, Benjamin William Dudley, John Kearny Rogers, to speak only of the surgeons, who brought with them the English tradition obtained from various sources.

Wright Post was fortunate enough to live with John Sheldon in Great Queen Street, London, from 1784 to 1786. Sheldon at this time had just been elected professor of anatomy at the Royal Society of Arts. He was one of the great teachers in the Hunterian school of medicine, and while Wright Post was living with him he was elected surgeon to the Westminster Hospital. Post had, therefore, ample opportunities to learn clinical surgery as well as anatomy. Whether he profited morally as well as mentally we cannot now tell. John Sheldon was thirty years old and Wright Post was eighteen. Sheldon was engaged in the experimental embalming of dead bodies under the personal supervision of John Hunter, and when his mistress, whom he had picked up in Oxford Street, died of phthisis, he embalmed her and kept her in his bedroom until the lady he afterward married turned her out. She is now stowed away in a back room of the Royal College of Surgeons, where I often visit her. Wright Post, at any rate, does not seem to have alluded much in his after life to those days, for he is described as a grave and dignified man who never smiled.

Philip Syng Physick was even more fortunate than Wright Post. He came to London in 1789, three years after Wright Post had returned home, became a pupil of John Hunter and served as his house surgeon at St. George's Hospital. He was so thoroughly imbued with Hunter's teachings that he is rightly called the "father of American surgery," and for this reason I selected him from all other surgeons to be commemorated on that mace which was presented to the American College of Surgeons as a token of good-will. Physick afterward went to

Edinburgh, where he graduated doctor of medicine, but possibly our food or the Scotch climate did not agree with him, for we are told that he was "a cold, dyspeptic, pessimistic and unsociable man." It is a pleasure to discover that the large family of American surgeons who look upon him as their father have not inherited these traits.

Ephraim McDowell followed Philip Syng Physick at Edinburgh, where he was a pupil of John Bell in 1793. There is, I think, no doubt that John Hunter was the source of inspiration which led him to perform the ovariotomy in 1809, which was destined to become so celebrated in history. Twenty-three years previously John Hunter had stated publicly in the lectures which he delivered in the winter session 1786–87 that "Tapping is only a palliative cure (for ovarian hydatids) and a large trocar is required as the fluid is generally gelatinous and then only one cell is opened. If taken in the incipient stage they might be taken out as they generally render life disagreeable for a year or two and kill in the end. There is no reason why women should not bear spaying as well as other animals. It would be simply opening the cavity of the abdomen which we often do in healthy constitutions."

The gods loved the young Quaker, John Syng Dorsey. A man of singular promise, surgeon, artist and a pioneer in your surgical literature. A nephew of Physick, he spent a year in London and Paris, working at the Windmill School of Medicine under Everard Home, the brother-in-law of John Hunter. Returning to Philadelphia, in 1804, he tied successfully the external iliac artery, wrote a text-book of surgery in 1813, which passed through three editions and died of typhus before he was thirty-five years old.

> "Young Lycidas and hath not left his peer
> Who would not sing for Lycidas? dead ere his prime."

While McDowell was in Edinburgh, John Collins Warren was sent to London by his father, the professor of anatomy and surgery in the newly founded medical school of Harvard. He

arrived in 1799 as a pupil of William Cooper, surgeon to Guy's Hospital, but he was soon transferred to his nephew, Astley Cooper. Warren acted as a dresser at the Borough Hospitals under Astley Cooper for a year and afterward went to Edinburgh and thence to Paris, returning home in 1802. While he was in London, Sir Astley Cooper and Hey, of Leeds, were busy dissecting and describing the anatomy of the parts concerned in hernia, and it is worthy of note that Warren was one of the earliest surgeons in America to operate for the relief of strangulated hernia.

Valentine Mott, like J. C. Warren, had also the good fortune to be a pupil of Sir Astley Cooper when he visited London in 1806, and, like his master, he became a fearless surgeon and a good anatomist. You will remember how, when there was as yet no chloroform, he spent four hours in excising the clavicle for a huge growth, mentioning the arterial source of each vessel as he tied it. The patient lived afterward for very many years.

I think it probable that Astley Cooper's republican principles may have influenced the fathers of Warren and Mott to send their sons to him. Cooper had imbibed from his master, Henry Cline, those principles of "liberty, equality and fraternity" which nearly wrecked his career in London, for they almost prevented him from being elected to the staff of Guy's Hospital, and sometimes he used to say it gave him a queer feeling in his throat, "just as though a rope were round it."

William Gibson, of Baltimore, must have been as much at home in Europe as in this city, where we are meeting. He was a pupil of Wright Post and was sent to Edinburgh when quite a boy. Here he showed such artistic power that he soon attracted the attention of John and Charles Bell, who were equally skilful as artists and surgeons. He followed Charles Bell to London and there came under the influence of Astley Cooper. He was present at the battle of Corunna, in 1809, and in the following year he returned to Baltimore. Two years later he fought against us, and in 1815 he was helping on the French side at the Battle of Waterloo, so that by the time he had reached the age of twenty-seven he had had a very fine experience both in

civil and military surgery. He had succeeded Dr. Physick at the University of Pennsylvania, and in 1812 he tied the common iliac artery. In 1824 he published a text-book on surgery, which I have recently read with pleasure and some profit. It is charmingly written, and is much more modern in tone than any of the contemporary treatises.

Benjamin William Dudley was another carrier of European tradition to America, but, unlike the surgeons I have already mentioned, he had to rely on his own resources, for he had no wealthy father to help him. He was the son of a captain in the Revolutionary War, and, having a thirst for knowledge and a desire to see the world, he bought a cargo of flour at New Orleans, accompanied it across the Atlantic, sold it at Lisbon and Gibraltar, and then made his way through Spain to Paris. He spent four years in Europe, partly with Baron Larrey and partly with Astley Cooper and Abernethy. It is recorded that after his return home he cut 225 persons for stone successfully, using Cline's gorget.

John Kearny Rogers acted in like manner as an intermediary between England and America, devoting himself more especially to ophthalmic surgery, though he tied the left subclavian artery medial to the scalenus anticus. He was in London about the year 1817 and followed the surgical practice of Sir Astley Cooper and Sir Benjamin Brodie.

I cannot resist showing you John Peter Mettauer, who lived from 1787 until 1875, although he never visited Europe. He must have been a very fine surgeon, for he operated 800 times for cataract, and 400 patients he cut for stone. He did the first operation for the repair of a vesicovaginal fistula, and he was the first in America to cure a cleft palate, the operation being performed in 1827. Two years later he is said to have recognized that typhoid fever was a separate disease. He practised in Virginia, and excused himself from writing on the ground that he was never idle, yet in spite of this he married four times. It is told of him that he was never seen without his top hat; it was believed that he slept in it, and it appears certain that he was buried in it.

I think, gentlemen, you will see from the little sketch I have just given you how the tradition of British surgery traveled to you and how it influenced your early work. It came chiefly through London and Edinburgh, and in both places a sound knowledge of anatomy obtained by actual dissection was looked upon as the outstanding mark of a surgeon. Your professors of anatomy for many years held a highly distinguished position, and the professorship of surgery was often combined with that of anatomy. The work of John Hunter led your early surgeons to interest themselves in the ligature of the larger arteries. They had many opportunities of practice when syphilis was badly treated and wounds with cutting instruments were not uncommon, so that they left the world a remarkable record of results both in their successful and unsuccessful cases. Sir Astley Cooper was working hard at the subject of fractures, dislocations and hernia when your younger surgeons came under his influence, and they transported his enthusiasm to your country. These surgical emergencies were constantly arising. You soon made their treatment your own and you greatly advanced our knowledge. Your field of surgery then lay fallow for a time, civil discord prevented any advance and you fell a little behind. You have more than made up for lost time in recent years, and we are again your debtors, especially in connection with the surgery of the abdomen, both in men and in women, until at the present time there are a number of American surgeons who in originality, resourcefulness, skill and wide experience have no superiors and few equals; but you seem to me to be passing from one extreme to the other. Not long ago you were family doctors first and surgeons afterward; now you appear to be becoming specialists first and general surgeons afterward. It should surely be the other way round—you ought to be general surgeons first. It is no doubt easier to make money as a specialist and in larger amounts, but it is not good for surgery as a profession to take this view. It is making a trade of one's work. The real surgeon, like the artist, works for the joy of working, and not that he may leave his posterity with so much money that they run a chance of becoming decadent.

SURGERY OF THE RIGHT HALF OF THE COLON

By JOHN B. DEAVER, M.D.
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The right half of the colon differs embryologically from that of the left half. The proximal colon is developed from the midgut. It comprises that portion of the bowel which extends from the ileocecal junction to the splenic flexure. This portion of the bowel, especially the cecum, the ascending colon and the hepatic flexure, is a frequent site of disease.

Within recent years much has been written on the subject of carcinoma of the large bowel. However, a careful perusal of the literature will show that the attention of the surgeons has been focussed chiefly on that portion of the colon which is most frequently affected—that is, the sigmoid, rectosigmoid and rectum.

It is very difficult to obtain a careful comparison of the occurrence of cancer of the colon to that of other organs. In the collected statistics of Hoffman he gives as the cause of death in the registration area of the United States, in the years from 1908 to 1912, cancer of the stomach and liver, 80,316; of the buccal cavity, 7716; of the skin, 7595; of the female generative organs, 30,907; of the female breast, 18,884; of the peritoneum, intestines and rectum, 25,644; of other or unspecified organs, 31,559. It is interesting to note that when this is considered at the rate per 100,000 of population, 7.7 per cent of the male and 11.3 per cent of the female deaths are due to cancer of the peritoneum, intestines and rectum. It is of further interest to note that while in 1900 only 5.7 per cent of the deaths per 100,000 were due to cancer of the peritoneum, intestines and rectum; in 1913

this had risen to 10.5 per cent. It may be that this increase is due to more careful diagnosis. On the other hand, Hoffman points out that "there is no evidence that the disease groups to which cancer might erroneously have been assigned have materially decreased, if at all, coincident with the gradual rise in the cancer death-rate."

When we come to consider the frequency of cancer of the large bowel with that of the small bowel we find the following statistics:

At the Pathological Institute of Vienna, of 343 intestinal carcinomata which came to autopsy, 164 were in the colon and 162 in the rectum, while only 7 were in the duodenum and 11 in the ileum. In Schleip's collected series of 542 intestinal carcinomata 257 were in the rectum, 259 in the colon, 20 in the duodenum and 16 in the ileum. In Brill's series of 3563 intestinal tumors, 97.5 per cent were in the colon, appendix or rectum. In Herman's collection of 20,544 cancer cases, 1706 had a lesion in the large intestine, 1204 in the rectum and only 20 in the small bowel.

An analysis of numerous statistics made in order to determine the frequency of cancer of the various anatomical parts of the large bowel gives the following figures: In Mummery's collection of 188 cases of cancer of the large intestine, exclusive of the rectum, 103 had carcinoma of the sigmoid flexure, 6 of the descending colon, 12 of the splenic flexure, 17 of the transverse colon, 3 of the hepatic flexure, 6 of the ascending colon and 41 of the cecum. At the Mayo Clinic, from January 2, 1915, to December, 1922, of 359 cases of cancer of the colon 71 were of the cecum, 44 of the ascending colon, 28 of the hepatic flexure, 50 of the transverse colon, 23 of the splenic flexure, 39 of the descending colon and 104 of the sigmoid flexure.

Of 511 deaths from carcinoma of the large bowel reported by Azeman, Maydl, Müller and Nothnagle, 35 were from cancer of the cecum (6.8 per cent), 131 from cancer of the colon (25.6 per cent), 83 from cancer of the sigmoid (16.2 per cent) and 262 from cancer of the rectum (51.2 per cent).

Erdmann, in a series of 108 cases of carcinoma of the large bowel, reported 39 in the rectosigmoid, 4 in the left one-quarter of the transverse colon and the splenic flexure, 12 in the hepatic flexure and the right three-quarters of the transverse colon; the remainder of the cases he did not localize.

It is generally agreed that if we consider the question of carcinoma of the colon, exclusive of the rectum, about 36 per cent occur in the sigmoid and 25 per cent in the cecum, the transverse colon and the splenic flexure; the hepatic flexure and the ascending and descending colon being accountable for the rest in about that order of frequency. In the Lankenau Hospital Clinic, Philadelphia, among the last 60 cases requiring surgery (exclusive of fecal fistula and non-malignant obstruction), the right half of the colon was involved 15 times, or in 25 per cent of the cases.

Before taking up the various clinical aspects of the subject it is important to discuss the factors which may predispose to the development of carcinoma in this portion of the bowel, especially if we accept the statistics which show a steady increase in the incidence of the disease. If we can recognize the conditions which precede and those which produce cancer of the colon we will have taken a long stride forward in its control and prevention.

Trauma to the bowel, as elsewhere, is a common predisposing factor in the production of cancer. Added to this, we have the various degenerative changes which occur in the body tissues, which have been ascribed merely to age and again to intestinal putrefactive processes, and still again to syphilis. The most important factor, however, is unknown. It may be organismal or not. It may be, however, that this unknown factor acts in conjunction with one or the other of the known factors and the absence or presence of one or the other decides for or against malignant degeneration.

The right half of the bowel affords an excellent illustration of the manner in which traumatism produces ulceration and carcinoma. I am one of those who believe that chronic ulcera-

tion plus traumatism is all that is necessary to produce malignant change. The lesions of the tongue are also excellent examples of this. The cecum and hepatic flexure can be compared with the sites in the esophagus and the cardiac and pyloric portions of the stomach where cancer develops in those locations subject to constant injury. Most of us will agree that if the irritant is allowed to exert its baneful influence over a sufficient period of time malignancy may result.

In the large intestine stasis is very frequent. Marked ptosis of the transverse colon, with a resulting increase in back pressure in the right half of the colon, affords sufficient trauma. Associated with this we frequently find the mucous membrane showing varying grades of inflammation.

When we come to consider the difference in frequency of cancer of the right half and of the left half of the bowel it should be borne in mind that the material in the large intestine varies in consistency, for while in the cecum and ascending colon it is still liquid or pasty, in the iliac and pelvic colon it is hard and firm. The greater frequency of cancer of the cecum in comparison with that of the splenic flexure may be due to the fact that the caput of the colon is similar to a reservoir.

It is unfortunate, though true, that the surgeon is still not consulted until either the tumor is grossly palpable or the patient is suffering from an acute or chronic obstruction. Although outwardly these may be the first signs, a careful history will show that the patient has had either pain, cramp-like in character, or perhaps only discomfort giving a sense of some intestinal derangement. At times the patient will localize accurately the area in which exaggerated peristalsis is attempting to overcome a partial obstruction. Long before the tumor is palpable, borborygmi may have been noticed. Irregularity in fecal evacuations are fairly constant, and if the lesion is in the right half of the colon this may be in the form of diarrhea. Sir Berkeley Moynihan has said that in his cases "It was very rare to find constipation as a symptom of a growth in the right colon and rare to find it absent in a growth of the left colon."

The constituents of the feces may also be altered; blood is probably always present and can be found if repeated examinations are made. The excess of mucus associated with colonic tumors is due to an inflammation of the mucosa above the growth. An early anemia is especially significant of neoplasms of the right half of the colon, Moynihan finding it in 50 per cent of the patients who had tumors of the ascending colon and in 20 per cent of all colonic growths. In fact, the blood picture may simulate that of pernicious anemia.

The tumor at first is small and may not be palpable unless the individual is thin. At times the tumor felt is not the actual growth, but is an accumulation of feces behind it. Occasionally visible peristalsis is present, or if this cannot be seen, by careful palpation the bowel may be felt to distend and then slowly to relax under the hand as the contents of the colonic lumen pass the obstruction.

Carson in his analysis of 111 colonic cancers found that 68 (62 per cent) were in the iliac or pelvic colon, of which 50 per cent were obstructed; 18 (16 per cent) were in the cecum or ascending colon, of which 33 per cent were obstructed; 9 (8 per cent) were in the transverse colon, of which 6 (66 per cent) were obstructed; 9 (8 per cent) were in the descending colon, of which 7 (77.7 per cent) were obstructed; 6 (5.4 per cent) were in the splenic flexure, of which all were obstructed. This shows that the growths of the right half of the colon are second in frequency to those of the ileopelvic colon and are much less liable than any of the tumors of the large bowel to become acutely obstructed, while tumors of the splenic flexure and descending colon are obstructed in 90 per cent of the cases.

The patients are usually in advanced life, but numerous cases have been reported between the ages of twenty and thirty years, so that youth does not exclude the possibility of cancer of the colon. Statistics show that the male is more commonly affected than the female, the proportion being from 2 to 1 to 3 to 1.

I feel that it is a mistake to rely too much on the *x*-ray diag-

nosis. In fact, where the clinical diagnosis has been clear the *x*-ray findings have frequently been negative. The surgeon who waits for the *x*-ray to give positive findings may miss the favorable time when radical operation may effect a cure.

The chief conditions which are to be differentiated from cancer of the right half of the colon are tuberculosis and actinomycosis, bands and adhesions causing partial obstruction, chronic appendicitis and the occasional presence of diverticula. Since these conditions are primarily surgical, it would seem that accurate diagnosis is only of academic interest, while procrastination is accompanied by serious hazards.

The colonic cancer is usually slow-growing. It remains restricted to the intestinal wall for a long time. Sampson Handley, some years ago, shocked the surgical profession when he announced that he had found permeation of cancer cells six inches from an apparently localized growth. However, I think he later partly denied the assertion. There can be no doubt that for a long time adenocarcinomata of the right colon are confined to the mucosa and submucosa. During the process of growth the cells spread by (*a*) direct extension, (*b*) through the venous system and (*c*) through the lymphatic system. Hausmann's statistics are of the greatest importance. Thus in 112 autopsies on cases of cancer of the colon, in 21 the disease had spread beyond the bowel and become generalized; in 36 only the primary lymphatic glands were enlarged; in 55 cases the disease was limited to the bowel. These figures compiled from patients dying of cancer of the colon showed that 50 per cent of them had died without any sign of the growth outside of the bowel. This low-grade malignancy of colon cancers should afford the surgeon an opportunity for excellent results if the cases are operated on before contiguous structures are involved.

In 359 cases from the Mayo Clinic 140 (about 39 per cent) had metastasis to lymph nodes or to other organs. In these the primary growth was in the cecum in 34 cases, in the transverse colon in 25 and in the sigmoid in 36. Stated in percentages,

metastasis had occurred in 48 per cent of the cecal growths; 50 per cent of those in the transverse colon, 35 per cent of the sigmoid cancers and 31 per cent of those situated elsewhere.

Only one who has operated on many of these cases can understand the varying malignancy of colonic tumors. In no other part of the body is there a wider variation in the degree of malignancy. The virulence of bowel cancer depends more upon the seed than the soil. The colloid cancer is most malignant, the scirrhous cancer next and the fungating type the least malignant. Generally the growth is slow, and for a long time the disease is limited to the bowel and the lymphatic glands are invaded late. The duration of life after simple colostomy in inoperable growths is often prolonged as compared with that of the inoperable carcinomata.

With such a state of affairs it is interesting to inquire into the number of patients presenting themselves for treatment who are suitable for a radical or a palliative operation. Statistics must be accepted with some reservation, since surgeons vary in their opinions as to radical operability, and, furthermore, the older statistics show a lower operability than the more recent ones. This is ideally illustrated in the Mayo Clinic reports, where, during the years 1910–1913, 51 per cent of the cases of cancer of the rectum had the radical operation, while in the succeeding two years (1913–1915) 71.8 per cent of the cases were deemed suitable for radical intervention. Accurate statistics on the operability of tumors of the right half of the colon are not available, since what one surgeon considers suitable for radical treatment may be considered unsuitable by another.

Again, I quote from the Mayo Clinic: In the 359 cases of cancer of the colon before mentioned 125 were in the cecum and ascending colon and over two-thirds were found suitable for radical resection. McGlannan, in a series of 98 cases of cancer of the colon, found that 61 gave a history of an obstruction of some sort before operation. The suitability of the lesion for radical operation depends (1) upon the location of the area, (2) upon the extent of the lesion and the metastasis

and (3) upon the associated local or general disease. Under this heading I also consider obesity, since this is a disease, and there can be no doubt that the mortality is higher in obese patients.

There are definite contraindications to the radical operation. If the lymphatic glands at a distance from the area to be excised are invaded by cancer cells I doubt the advisability of the radical operation. The removal of the lymph glands in the ileocecal region is comparatively simple, since these glands lie along the ileocolic and the right colic arteries. Enlarged glands are not necessarily cancerous. It must be remembered that infection, which invariably accompanies cancer, may be the cause of glandular enlargement. Jameson and Dobson have carefully worked out the lymphatic drainage of the colon. They classify the glands into four groups: (1) The epicolic glands, which lie on the bowel wall and which drain into the next two groups; (2) the paracolic glands, which lie in the mesentery along vascular arches close to the gut; (3) the intermediate glands, lying on the arterial branches between the vascular arches and the main trunk; (4) the main glands, situated around the colic arteries close to their origin, and into which all of the foregoing drain. On the other hand, growths in the hepatic flexure and the right half of the transverse colon metastasize to glands about the pancreas and along the sides of the aorta. The radical removal of the carcinoma in this latter region is therefore much more difficult and is attended by greater hazards. All of us have seen cases in which only local excision of the growth was practised and the patient lived comfortably for a number of years. Paul, of England, emphasized this in 1912, when he said, "Many cases having the minute structure of cancer have not recurred, though known to have been removed within an insufficient margin of safety." He also said, "Malignant disease of the bowel is very rarely removed during the early stage, yet the percentage of cures is remarkably good." Further contraindications to the radical operation are

deep invasion of the muscles of the posterior abdominal wall and extension to the parietal peritoneum. Moynihan believes that it is feasible to remove the invaded muscles without adding to the gravity of the operation; thus the contraindications also vary according to the surgeon. William J. Mayo has shown that with increasing boldness and skill on the part of the surgeon in attacking growths of the large bowel the percentage of patients cured has been greatly increased, even though the operative mortality has been raised.

Whether the operation should be done by the one-, two- or several-stage method is a matter of some controversy. The pioneers in this field resorted to the use of a temporary colostomy, done either at the time of the radical operation or as the first stage of this operation. As surgery progressed, and as infection and wound-healing were better understood, the one-stage operation took precedence over the two-stage procedure. I believe that in the ileocecal region conditions still favor the one-stage operation. The contents of the bowel are fluid and their infective power is not nearly so great as in the left half of the colon. The ileum is nearly completely covered with peritoneum, and the peritoneum of the large bowel is sufficient to give adequate serous approximation. Resection of the right half of the colon is therefore safer and easier than resection of the left half. To these factors can be added the greater mobility of the right half of the colon and the fact that its blood supply is more constant than that of the left half. Judd and Rankin have suggested ileostomy in all resections of the colon. This, they say, serves as a "safety valve." The only indication that I can see for the two-stage operation for cancer in the right half of the colon is in cases suffering from acute obstruction when first seen.

Cumston and Vanderveer, in 1902, reported 83 cases of excision of the cecum for cancer. Of 73 of these in which accurate statistics were obtained 30 died under the operation and 43 recovered. McGlannan, in 1914, reported 32 cases of cancer of

the cecum, 10 of whom were cured, 18 of whom were dead and 4 of whom were living, but inoperable. He also reported 17 cases of cancer of the ascending colon and hepatic flexure, 1 of whom was cured, 15 of whom were dead and 1 living and inoperable. How long after operation the death occurred or how long after operation the "cures" were reported was not stated in his report.

In McGlannan's cases a two-stage operation was done when acute obstruction was present and a one-stage operation in the other cases.

Other conditions, which are occasionally met with in the right half of the colon requiring operation, are actinomycosis and tuberculosis. These practically always occur in the cecum. Waring reported 7 cases of actinomycosis of the cecum or appendix, and to these have been added 14 cases by Brogen, 10 of which originated in the ileocecal region. Two forms of the disease are seen: The acute form, which resembles appendicitis; and the chronic form, which is insidious in onset and associated with slight, indefinite pain in the right lower abdomen. Occasionally there is no pain, a mass being the first sign of the disease. In both types there is marked loss of weight and strength, associated with a pronounced anemia, the skin over the mass becomes bluish-red and sinuses occur frequently. Constipation and not diarrhea is the rule. The difference from carcinoma is that the latter is slower in growth, the anemia is not so pronounced, diarrhea is the rule and blood in the stools is more constant. The carcinomatous mass is more movable, causes no discoloration of the skin and there is no tendency to invade the anterior abdominal wall or form sinuses. It is usually impossible to remove all the diseased tissues at operation because of the diffuse infiltration. The most that can be done is to drain the abscesses, curette the sinuses and irrigate the area. At the same time large doses of potassium or sodium iodide should be given by mouth and the area in the region of the infection treated by the *x*-ray.

No.	Sex.	Age.	Diagnosis.	Operation.	Pathology.	Operative result.	End-result.
1415–23	M.	65	Carcinoma of cecum	Exploratory, Mar. 9	Large mass (carcinoma) in right iliac fossa; metastasis to bladder wall, mesentery of small bowel; carcinomatous mass near pyloric end of stomach; primary growth undetermined	Recovery	Died 4 months after discharge.
2132–23	M.	49	Carcinoma of cecum	Resection, ileocolostomy, July 4	Hard mass, size of lemon, in cecum; piece of omentum adherent to cecum excised; terminal ileum and cecum resected; ileocolostomy; lateral anastomosis	Recovery	5 months (444),* feeling occasional pain in scar; gain of 26 pounds since discharge.
549–22	M.	58	Carcinoma of cecum	Ileocolostomy, Feb. 23 hemorrhoidectomy, Mar. 22	Mass in cecum extending to hepatic flexure; gall-bladder adherent to mass; stones palpated in gall-bladder; large external hemorrhoids	Recovery	Failed to reply to various inquiries.
1804–21	M.	72	Carcinoma of cecum and descending colon	Ileosigmoidostomy, July 12	Diffuse carcinoma involving cecum; small carcinoma involving descending colon	Recovery	31 months (420); did well for 14 months, then began to fail; now has constant diffuse pain in abdomen; gas passes with difficulty; colon distended; sleep and appetite good.
M. F.	M.	54	Carcinoma of cecum	Ileocolostomy; excision of dermoid cyst of transverse mesocolon, Sept. 19, 1919	Mass, size of hen's egg, removed from mesocolon; cecum tied down by mass at ileocecal valve; carcinoma	Recovery	No follow-up.†
31–24	F.	39	Carcinoma of hepatic flexure	Ileocolostomy, Jan. 4	Large growth on hepatic flexure, involving ascending colon; inoperable, ileocolostomy; lateral anastomosis	Recovery	
2654–22	M.	55	Carcinoma of ascending colon	Resection of colon, ileocolostomy, Sept. 11	Large, hard mass, size of orange, on ascending colon; metastasis to mesenteric lymph nodes; 15 cm. of ileum and all of cecum and ascending colon, with part of transverse colon resected; ileocolostomy; lateral anastomosis	Recovery	Died 12 months after operation.
2056–21	M.	48	Carcinoma of ascending colon	Resection and ileocolostomy, July 7	Annular carcinoma, size of hen's egg, on ascending colon, about 12.5 cm. from cecum; resection of bowel; ileocolostomy	Recovery	Did well for 11 months, then 2 small nodes appeared in incision; gradual decline; died April 22, 1922 (22 months after operation).

243–21	F.	50	Carcinoma of hepatic flexure	Ileosigmoidostomy, Jan. 4	Large, hard, nodular mass adherent to under surface of liver, involving head of pancreas; neoplasm of hepatic flexure; omentum adherent to cecum, pulling down transverse colon; ileocolostomy	Recovery	Failed to reply to inquiries.
M. F.	M.	66	Carcinoma of ascending colon	Ileocolostomy, Aug. 18, 1919	Neoplasm on ascending colon; preliminary ileocolostomy; secondary operation not performed	Recovery	No follow-up.*
S. E.	M.	49	Carcinoma of ascending colon	Resection and ileocolostomy, April 3, 1919	Small intestine distended; descending colon distended as far as hepatic flexure; obstruction at hepaticflexure; ileocolostomy at middle of transverse colon	Recovery	No follow-up.*
3612–23	M.	37	Tuberculosis of cecum	Cecectomy; ileocolostomy, Nov. 14	Great omentum adherent to cecum and parietal peritoneum; wound enlarged; ileocolostomy	Recovery	4 mos. (444); the wound healed in 2 months; no complaints; bowels regular; appetite good.
857–23	F.	35	Tuberculosis of cecum and ascending colon; pregnancy	Exploratory, Mar. 20	Extensive process involving cecum and ascending colon; uterus contains 4 months' fetus	Recovery	May 1, 1922, hysterotomy; 7 months improved; 18 months operated elsewhere; release of the adhesions of cecum and obstruction of cecum and descending colon; gaining strength (434).
L. G.	F.	22	Tuberculosis of cecum	Ileocolostomy, Jan. 16, 1919	Many adhesions about the ascending colon too dense for separation; either tuberculosis or carcinoma	Recovery	No follow-up.*
2331–22	F.	34	Tuberculosis of cecum	Ileojejunostomy, jejunojejunostomy, Aug. 1	Point of obstruction, about 5 cm. long, between a distended and collapsed portion of small bowel; lateral anastomosis above distended portion; second anastomosis lower down; entire jejunum and part of ileum infiltrated by what appeared to be a tuberculous process	Recovery	Died 1 month after discharge.

* Patients are rated according to results.
† Follow-up system inaugurated in 1920. Cases before that time not followed for this study.

Tuberculosis at the ileocecal region is not uncommon. Fenwick and Dodwell report 85 per cent of the cases of intestinal tuberculosis as involving the ileocecal region. The disease may be localized or disseminated. Two forms are described—the ulcerative and the hypertrophic. The ulcerative form may simulate actinomycosis while the hypertrophic form simulates carcinoma or occasionally appendicitis. The hypertrophic form is by far the more frequent, and is characterized by anorexia, intestinal upsets and pain in the right iliac fossa, diarrhea and constipation, with occasional attacks of partial or complete obstruction. The tumor is hard and somewhat nodular and by palpation may be indistinguishable from carcinoma. Tuberculosis more frequently occurs below the cancer age. The treatment is entirely surgical, complete removal of the diseased area with a side-to-side or end-to-side anastomosis being done.

SUMMARY OF OPERATIONS ON THE RIGHT HALF OF THE COLON

Fifteen Cases among Last Sixty Cases of Surgery of the Colon (Exclusive of Fecal Fistula and Obstruction)

Diagnosis.	No.	Operation, exploratory.	Resection and ileocolostomy.	Ileosigmoidostomy.	Ileocolostomy.	Ileojejunostomy.	Total.
Carcinoma of cecum . . .	5	1	1	1	2	..	5
Carcinoma of hepatic flexure	2	..	..	1	1	..	2
Carcinoma of ascending colon	4	..	3	..	1	..	4
Tuberculosis of cecum . .	4	1	2	..	..	1	4
	15	2	6	2	4	1	15

Another lesion of this area requiring surgery is appendiceal fecal fistula. The complication of appendiceal fecal fistula is

confined to the case presenting perforation, abscess and drainage. In the last 4063 cases of appendicitis at the Lankenau Hospital Clinic, Philadelphia, 200 were followed by fecal fistula, an incidence of approximately 5 per cent. In every instance pus was present, in more or less amounts, requiring drainage, but in the vast majority of cases some ulceration of the cecum or terminal ileum was noted at the time of the primary operation. The cases which had perforated at the base of the appendix displayed the greatest tendency toward the formation of a fistula. Of the 200 fistulas 74 (37 per cent) healed spontaneously, while 97 (48.5 per cent) required operative repair. The remainder, 29 (14.5 per cent), left the hospital without operation. No doubt a certain proportion of these closed spontaneously. The type of operation for the repair of the fecal fistula depended upon the conditions existent at the time of operation. In 60 per cent (120 cases) of the cases simple inversion of the fistulous opening by a purse-string linen suture reinforced by an additional suture line sufficed, with 3 deaths. In 15 per cent (30 cases) the condition of the bowel was such as to excite doubt as to its regenerative power in the presence of the fecal stream, so that an added ileocolostomy was performed, with 4 deaths. In 25 per cent (50 cases) there were either multiple fistulas or an opening so large as to preclude closure with maintenance of the lumen of the bowel, with 2 deaths. These required resection of the bowel, varying from a small portion of the cecum to resection of a foot or more of the terminal ileum with the cecum and ascending colon. Ileocolostomy was, of course, the last stage of the operation.

CARCINOMA OF THE COLON

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This report is based on a study of 67 cases of carcinoma of the colon, excluding the rectum, treated on the second surgical division of the Roosevelt Hospital since 1909.

The grave character of the disease, the high operative mortality, the frequency of recurrence after resection, the large percentage of cases too advanced to permit of removal when first seen all tend to make the outlook in these cases far from encouraging.

It is the object of this paper to try to demonstrate the fact that carcinoma of the colon compares favorably with malignant disease elsewhere in percentage of operability, and in favorable results, both immediate and late, following radical operation.

It is true that many cases first seek surgical advice when the disease is well advanced and beyond hope of surgical cure. Many are first seen in acute obstruction when the diagnosis can only be surmised and emergency colostomy or enterostomy must be resorted to as a life-saving measure. Some of these patients when questioned later give a history of increasing constipation or attacks of partial obstruction, which should have given warning leading to an earlier diagnosis; other attacks come suddenly in patients in apparent good health. A case now under my care illustrates this type:

A woman, aged fifty years, had always been well until January of this year, when she had an attack of cramp-like abdominal pain attributed to indiscretion in diet, which passed off under

treatment by catharsis in two or three days. There was no previous history of increasing constipation or abdominal pain, and after the attack the bowels moved normally until a second attack of pain and distention, occurring three weeks later, failed to respond to catharsis and enemata, and emergency cecostomy had to be performed for complete obstruction on February 6, 1924. Radical operation by the two-stage (Mikulicz) method, performed on March 8, revealed a large carcinoma at the splenic flexure, without glandular metastases or secondary deposits; the lumen of the gut at the growth was barely the size of a lead-pencil. She made a good operative recovery.

The first symptom to attract attention may be attacks of colicky pain with abdominal distention, and increasing constipation in the intervals.

Careful palpation, when the abdomen is flat, may reveal a mass, especially when it is located in the cecum, ascending or transverse colon, the lower end of the descending colon or the sigmoid. When at or near the hepatic or splenic flexures it may be difficult or impossible to locate, as it is also in obese or very muscular patients or in the presence of abdominal distention.

The discovery of a mass or tumor may be made before any obstructive symptoms have developed or other evidence of disease has appeared, or bleeding from the bowel may be the first warning. Bleeding may appear as a single large hemorrhage, as bleeding repeated in moderate amount or as occult blood in the stools.

In some cases, especially right-colon growths, severe secondary anemia, debility and loss of weight may precede local evidence of the disease.

Whatever the initial symptoms may be, a thorough diagnostic investigation should be made at once and the site of the growth located as nearly as possible.

When advanced obstruction is present, colostomy or enterostomy must precede this investigation, and it is often impossible or unwise to attempt to locate the growth by exploration when

this is done. It is far better to do a cecostomy or ileostomy on the right side than to jeopardize the patient by a difficult exploration in the presence of extreme distention.

When the distention is relieved the abdomen should be carefully palpated for a mass, digital examination of the rectum (a procedure too often omitted) and sigmoidoscopy should be done, and *x*-ray examination after barium enema, the most valuable of our newer diagnostic methods.

Great care must be taken in interpreting colon radiograms, as apparent filling defects occur from various causes and erroneous conclusions are not infrequent. Differential diagnosis must be made from many other conditions which cause obstruction or abdominal distention.

Obstruction from bands or kinks; from partial volvulus of redundant loops of sigmoid; from impacted feces in elderly people; distention from paralytic ileus in the course of systemic disease, *e. g.*, pneumonia, all are conditions in which we have seen errors of diagnosis made.

When the diagnosis has been established and the site of the growth located either by diagnostic examination or exploratory operation the plan of treatment must be determined.

In many cases the presence of a large, fixed growth; of local extensions or involved lymph nodes or of palpable nodules in the liver make it all too evident that palliative colostomy is the only operation to be considered.

In the massive advanced growths without much obstruction, it is often wise to omit even this procedure, to avoid the distress and annoyance of a fecal opening during the last few weeks of life.

Sometimes a short-circuit operation may be done for obstruction; or it may be justifiable to resect a large painful growth, even when nodules are present in the liver, especially when it can be done safely by the two-stage method, for the relief of pain and obstruction.

We have done this twice: once in a recent case still under treatment, a large growth at the splenic flexure, without lymph-

node involvement or local extension, but with multiple nodules in both lobes of the liver. The other case, a descending colon growth with liver involvement, recovered from the operation and was relieved of abdominal symptoms, but died seventy-three days later of cerebral metastases.

Pathologic Types. There are several gross pathologic types, of which the most common are the round ulcer and the scirrhous contracting growths.

The round ulcer gradually increases in size until if left alone it finally encircles the gut, contracting as it grows and producing, as a rule, marked constriction. The growth advances in the submucous layer beyond the limits of the ulcer; the edges are piled up and thickened, not generally undermined, and the base is covered with unhealthy granulations which bleed easily.

The cases vary in the proportions of cell mass to fibrous tissue, some being quite dense and hard, others spongy and cellular; the latter bleed most easily. While the rate of growth varies greatly, it is often slow, and glandular metastases and the development of secondary deposits in the liver are fortunately often long delayed.

It is the type which in the long run offers the best hope of successful eradication.

The scirrhous contracting type is also slow of growth, narrowing the gut lumen as the growth progresses, until finally attacks of partial obstruction give warning of its presence, or the narrowed opening may be suddenly plugged with a mass of hardened fecal matter and complete obstruction ensue, apparently out of a clear sky. While this type is also of relatively low malignancy, with late metastases, not infrequently exceptions to this rule occur in the form of liver nodules or retroperitoneal gland involvement.

The gut above the constriction may be much dilated and its wall thinned. Rupture above the constriction is possible, and we have seen it occur in the cecum in one of our cases, the result being a huge intraperitoneal fecal abscess and a fatal issue. In another case a contracting growth in the middle of the trans-

verse colon in which the obstruction had been relieved by an enterostomy in the small intestine there was absolute water-tight closure of the lumen of the colon at the time of its resection. Block resection of the colon with end-to-end suture was followed by spontaneous closure of the fecal opening. Death occurred, however, within a year, due to extensive metastases in the liver and the peritoneum.

When constrictions occur in the ascending colon or cecum the lower ileum undergoes hypertrophy and marked dilatation.

This often produces the symptom called stiffening of the bowel, or disappearing tumor. The lower ileum goes into a state of tonic contraction similar to that of the gravid uterus; a tense mass formed by the intestinal coils can be seen and felt, and if the hand is kept on the abdomen, in a few moments the spasm relaxes, the gut wall softens and the tumor disappears. This is simply a later phase of the visible peristalsis seen in these cases.

Perforation of the growth itself from deep ulceration, with pericolic abscess formation, occurred in 5 of our cases; in 1 case the perforation was plugged with an orange seed.

Drainage was done in 2 cases, a cecal growth in a woman, aged seventy-two years, and a descending colon growth in a man, aged forty-four years. Simple drainage of the abscess was followed by a fatal result in the cecal case in twenty-four hours. Drainage of the abscess and cecostomy, by death occurring on the sixth day in the second case.

A third case made an operative recovery after drainage of the abscess with a short-circuit operation.

Two cases died promptly of acute sepsis after radical resection, with contamination of the operative field from the perforation, both left colon cases.

The third type is the massive medullary growth, with early lymph-node involvement and metastases. These cases grow more rapidly and are prone to hemorrhage.

While attempts at radical resection are usually discouraging in this type, one of our cases, a man, aged twenty-nine years, the

youngest of the series, lived for three years and four months after operation, an extensive right colon resection, and had more than two years of active work in comfort as principal of a large public school. The growth was of the medullary type, with colloid formation; lymph-node involvement was quite extensive at the time of operation.

We have had 67 cases of carcinoma of the colon on the second surgical division of the Roosevelt Hospital since 1909.

Of these cases, 37 (55 per cent) have been treated by radical resection; 19 right-colon growths and 18 left-colon growths.

Of the right-colon growths, 17 were resected by the one-stage method with immediate suture, 13 side-to-side, 3 end-to-end and 1 by the end-to-side method.

Fifteen of the 17 recovered and 2 died; 1 recent case, end-to-side suture, from obstruction at the site of anastomosis, in spite of a secondary enterostomy; the other after the wound had healed, and he was out of bed on the thirty-third day after operation, from cardiac decompensation. The operative mortality of this group was 11.7 per cent, or, excluding the cardiac death, 5.8 per cent.

Two right-colon cases were resected by the two-stage method; 1 recovered and 1 died.

Of the 18 left-colon resections, 8 were done by the one-stage resection, with immediate suture, with 3 deaths, all due to acute sepsis, a mortality of 37.5 per cent.

Ten were done by the two-stage method, with 3 deaths, a mortality of 30 per cent—2 were due to acute sepsis and 1 to late cerebral metastases. The operative mortality of the series, 37 cases with 9 deaths, was 24.3 per cent.

A study of these statistics illustrates the fact that resection of the left colon is attended by a higher mortality than resection of the right colon, in which a one-stage operation, with immediate suture is a relatively safe procedure; that the difference is due to the higher infection potency of the left-colon content, a fact long recognized, and also to the greater difficulty of securing healing without leakage in suture of the left colon.

These facts are leading us more and more to resort to the two-stage procedure of Mikulicz in left-colon growths, and we believe that this procedure will greatly reduce the mortality in this group in spite of the fact that the results in the short series quoted do not demonstrate this. The real operative mortality in the 10 cases so treated, excluding the late death (seventy-third day) from cerebral metastasis, was 20 per cent rather than 30 per cent, while the mortality, all due to acute sepsis, of the one-stage method, was 37.5 per cent.

Three of our right-colon cases had severe secondary anemia when first seen, with the following blood counts: Hemoglobin, 35 per cent; red blood cells, 2,914,000. Hemoglobin, 40 per cent; red blood cells, 3,368,000. Hemoglobin, 46 per cent; red blood cells, 4,000,000. All were transfused first and a one-stage entero-colectomy performed by lateral suture. All made excellent recoveries, and are now well at thirteen years, nine years and three years and ten months respectively.

Right-colon growths, even without gross hemorrhage, are prone to produce advanced degrees of secondary anemia with great debility, the exact cause of which is somewhat obscure. They offer, in spite of this, some of the best prospects for a radical cure, as several cases in our series illustrate.

We quite appreciate that the above results are not brilliant and must turn to late results for a more hopeful view of the prognosis.

Of the 28 cases which recovered from radical resections, 15 are alive and well at the present time without recurrence; 1 is alive with recurrence at four years and four months after operation. One was well six and a half years and then lost to our follow-up in 1916; 1 died eight years after operation, free from recurrence, following an operation for septic cholangitis; 1 two and a half years postoperative, free from recurrence, following an extensive resection for persistent colostomy opening, a remarkable and complicated case, to which I will refer later.

Seventeen cases out of 28, therefore, are well or died free from recurrence, after long periods, 60.7 per cent. One lived six and

a half years and was lost track of, raising the percentage to 64 per cent. One is well four years, and four months, with recurrence.

Of the other late deaths, 1 followed six months after a second radical section for local recurrence at the ileocolic junction five years after the first operation. One was well eighteen months, developed recurrence and died three years and two months after the primary operation. Seven died of recurrent disease at from eight to eighteen months after operation.

The cases now alive and well are at the following periods after operation: Fifteen years, 1; thirteen years, 2; nine years, 1; four years, 3; two and a half to three years, 2; one year and ten months, 1; one year and five months, 1; seven months, 1; less than four months, 3.

It is the study of these late results which gives the grain of comfort in the study of cancers of the colon, results which compare very favorably with the results of cancer removal in other parts of the body.

Some of the individual cases present features worthy of mention:

A man, aged seventy-five years, with a large carcinoma at the splenic flexure and commencing obstruction, was resected by the two-stage Mikulicz method, made a good recovery and is now well and free from recurrence at one year and ten months postoperation. He spent all last summer traveling in Europe. Nineteen years before operation a carcinoma of the sigmoid had been resected successfully by another surgeon and the sigmoid and lower descending colon were entirely free from recurrent growth.

A woman, aged sixty-five years, was operated upon for a transverse colon growth quite adherent posteriorly to a retroperitoneal mass of glands, and was thought to be inoperable. A short-circuit operation was done between the transverse colon distal to the tumor and the ascending colon. Leakage occurred and a wide-open fecal fistula developed. After a period of three

weeks, in which death was expected at any hour, she began to improve; later an evaginated protrusion of gut, bearing at its apex a round ulcer type of carcinoma, appeared in the fistula, and finally protruded so far that the growth, with a good length of telescoped gut, was cut off and the ends sutured together. Recovery followed, but the fecal opening persisted; two and a half years later, after insistent demands on the part of the patient, extensive resection was done for the cure of the fistula, followed by death from peritonitis on the sixth day. There was no recurrence of the disease. The retroperitoneal invasion noted at the primary operation was evidently an inflammatory mass.

This instructive and disappointing case illustrates the well-known fact that adjacent infiltration and gland involvement, associated with ulcerated growths, may be inflammatory and not malignant.

SUMMARY. 1. Cancer of the colon, excluding the rectum, offers a relatively high rate of operability and a percentage of radical cures which compares favorably with that of malignant disease in other organs.

2. Growths of the cecum and right colon, including the right part of the transverse colon, are suitable for a one-stage resection with immediate anastomosis by suture. The operative mortality should be relatively low.

3. Left-colon growths are more safely resected by the two-stage Mikulicz method.

DIVERTICULITIS OF THE COLON

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Diverticulitis of the colon deserves repeated consideration because of the frequency with which it is encountered and the severity of the symptoms manifested in some of the cases. The treatment for the condition has not been definitely standardized, and it may be difficult to decide between operative procedures and less radical measures.

Diverticulitis was first described by Virchow in 1853, although apparently very little was known about it for many years afterward. Graser, in 1898, described a formation in the lower colon which he believed to be an acquired type of diverticulum. Fisher, in 1899, also described the condition, but the first comprehensive article affording a study of the etiology and classification of the several types was that by Beer in 1904. In 1907 Mayo, Giffin and Wilson reported the first series of cases (5) in which operation had been performed. The cases were studied from the standpoint of the clinical features, histologic changes in the tissues and the results of surgical treatment. Moynihan's paper on mimicry of cancer of the colon appeared the same year. Since then a number of detailed reports have appeared in the English literature by Telling, Drummond, Mummery, Hartwell and Cecil, Brewer, Rodgers and others.

Although postmortem statistics would indicate that diverticulitis occurs rarely, clinical experience, especially in the last decade, would indicate that it occurs often. In the Clinic during the last year one-third of the positive *x*-ray examinations made of the colon disclosed diverticulitis or diverticulosis. Diver-

ticula may be found in any part of the gastrointestinal tract from the esophagus to the rectum, but the condition which we speak of as diverticulitis occurs almost altogether in the sigmoid. Protrusions are fairly common in the right, transverse and descending colon, although they do not usually cause symptoms, probably because inflammation seldom occurs in diverticula in these areas. All diverticula of the sigmoid do not result in inflammation or manifest clinical signs; nevertheless, the sigmoid as a focus for diverticulitis is characteristic. In no other part of the colon do we find the bowel studded with rows of small saccules and the curious inflammatory thickening of the mesentery and other structures. Diverticulosis, or symptomless diverticula, occurs in any part of the colon. Diverticula of the sigmoid are more likely to become inflamed because the hardened fecal content of this part of the colon probably makes greater pressure within the sigmoid than in the other parts of the colon, and the fecal matter pressed into these saccules results in irritation and inflammation.

Etiology. Diverticulitis is a disease of middle life and old age. The average age of patients is about fifty-five years. The condition has been reported as occurring at the age of eighteen years. Two of Mummery's patients were twenty-one and twenty-three years, respectively. The youngest patient in our series was a boy, aged fifteen years, and the youngest on record is seven years (Ashhurst). The condition in this case was, we believe, verified by operation. The fact that diverticula rarely occur in young persons would indicate that they are not congenital, although this point has been widely discussed. Undoubtedly there is a congenital predisposition, possibly because of inherent weakness of the musculature. They are more common in the male, and in our experience are more common in fleshy persons. Constipation was one of the chief complaints in 63 per cent of our cases. In our review of these cases it did not appear that obstruction, either from stricture or adhesions, is a factor in the development of the diverticula. In most instances there was no demonstrable narrowing of the

lumen of the colon below the site of the diverticulum, and, furthermore, it is rather unusual to find diverticula in a colon which is markedly obstructed, so that while pressure must be considered a factor in the cause, some other factor must be responsible.

Hansemann and Klebs first called attention to the fact that the protrusions were closely related to the bloodvessels, entering and leaving the intestine along the mesenteric border. However, not all protrusions bear this relationship to vessels, and this explanation cannot, therefore, be applied to all cases. The part played by pressure and constipation, the bloodvessels and the congenital weakness of the wall of the colon in the production of these diverticula has been widely discussed, but most observers agree that a change takes place in the resistance powers of the wall. This change is probably slow and does not result in true diverticula until middle life. The inherent weakness, combined with the pressure of feces and gases in the intestine, is undoubtedly responsible for the condition.

Early in their development diverticula are probably microscopic in size, and all of the protrusions are true diverticula and contain all of the coats of the wall of the colon. Very early the musculature disappears, although the mucous membrane remains undisturbed. In our experience they have not been large. They are more common on the lateral wall, near the mesenteric border of the colon; occasionally they have a definite relationship to the fat tags. Our observations during the last few years lead us to believe that in most instances diverticula do not become inflamed, and hence do not produce symptoms. Since *x*-ray examinations of the colon have become reliable, we are finding many cases of diverticula of the colon without clinical manifestations. We believe, as Spriggs does, that these cases should be called diverticulosis in contrast to the others in which inflammation has developed. Wilson called attention to the mode of development of the inflammation in cases of diverticulitis. In several of the specimens he examined there had apparently been an escape of bacterial irritants without the

epithelium of the sac showing inflammatory changes. Just outside the submucosa, and within the fat or subserosa, he found a diffuse infiltration with leukocytes and a marked increase of fibrous tissue. He called this condition peridiverticulitis, and also directed attention to the fact that symptoms arising from the condition would be from inflammation in the peritoneum and not in the colon or diverticulum. It is likely that peridiverticulitis is the condition represented by a rope-like sigmoid with extensive infiltration of the fat and mesentery.

Secondary Pathologic Changes. Undoubtedly the diverticulum is the primary condition. It is not necessarily progressive, as shown by the fact that we have observed a great many patients for several years without discovering signs of change in the condition. The first change that occurs in the diverticulum is inflammation. We have recently been particularly interested in a study of the *x*-ray findings in these cases, with regard to the marked tendency to spasm of the colon. Spasm of the colon is, of course, a common occurrence, but the frequency with which it occurs in cases of diverticulitis is significant. Inflammatory changes in a diverticulum are practically identical with the changes which may occur in the appendix. Diverticulitis has been called the left-sided appendicitis of old people. When the pouch becomes inflamed and feces accumulate in it the muscle atrophies, the mucous membrane becomes thin and ulcerated, and the ulcer may perforate and result in a local abscess. This abscess may enlarge until it ruptures into the intestine or seals itself to the abdominal wall and perforates to the outside, or it may burst into the bladder, resulting in a colovesical fistula. We have seen many patients in whom an abscess had formed and ruptured into one of these viscera, but as yet have not observed the development of general peritonitis. In the 118 cases of diverticulitis of the sigmoid in which operation was performed at the Clinic, 14 localized abscesses were found in the peritoneal cavity, 3 in the abdominal wall, 1 in the wall of the bladder, 1 in the wall of the rectum and 1 in the liver. Besides these, 8 abscesses had perforated directly into

the bladder, so that feces and gas were passing through the urethra.

Usually the inflammatory process is chronic and fibrous tissue is deposited in and around the colon. Many adhesions form, and often a tumor composed of dense fibrous tissue results. In this tumor small abscesses and diverticula containing feces may often be found. The same condition usually occurs in several diverticula at once; the process is slow, probably taking several years. As a result of this chronic inflammation, stenosis of the sigmoid follows. The lumen of the sigmoid is reduced by the contraction of the fibrous tissue from the outside. The mucous membrane remains intact, so that in this respect a stricture from diverticulitis differs from a stricturing carcinoma. Blood was passed from the bowel in only 18 per cent of the cases of benign diverticulitis, while it was found in the stool in more than 47 per cent of the cases associated with carcinoma. A palpable tumor was present in 34 per cent of the benign cases and in 31 per cent of those associated with carcinoma. Constipation was one of the principal symptoms in about 60 per cent of the cases, but enough interference with the bowel to cause obstruction was present in 15 of the entire group of 137 cases. Many other patients had had obstructive attacks, as shown by the history, but there was no obstruction at the time they came for treatment.

It is likely that some of the patients with an indefinite history of chronic abdominal attacks have them as a result of inflammation in a solitary diverticulum. One of the problems which presents itself to the surgeon seems to be the decision of whether the symptoms are caused by the diverticulum. It has also been suggested that diverticulitis may act as a focus of infection and be the cause of remote symptoms. Rogers, writing on diverticulitis, reports 1 case of this kind which was entirely relieved by treatment for the diverticulitis. The mesentery usually becomes edematous and thickened and extends up over the sides of the colon. As a result of this swelling, and often after the inflammation in the mesentery has subsided, the colon

may be pulled into a distinct angulation, which may be the cause of obstructive attacks. As a result of this angulation it is often difficult to examine the colon satisfactorily with the sigmoidoscope, but when the characteristic condition is found it strongly indicates diverticulitis.

One of the interesting problems in the study of these cases is the association of carcinoma with diverticulitis. In the 118 cases in this series in which operation was performed there was no evidence of carcinoma. During the same interval we operated on 19 patients with diverticulitis of the sigmoid associated with carcinoma. Some years ago Wilson called attention to the manner in which diverticula of the sigmoid might become carcinomas, comparing the cases of carcinoma of the stomach developing secondary to ulcer of the stomach and carcinoma of the appendix developing secondary to inflammation in the appendix. We are unable to demonstrate whether this larger series of cases will bear out the contention that diverticulitis may result in cancer. In many of our patients in whom carcinoma and diverticulitis were associated there seemed to be no relationship between the two conditions, the carcinoma having apparently developed independent of the diverticulum. In some instances there were only one or two small diverticula remote from the carcinoma in the wall of the colon. It is probable that a patient with diverticulitis is no more likely to develop malignancy of the colon than one without it.

Clinical Manifestations. One of the striking features brought out in our study of these cases was the number in which diverticula had been diagnosed and in which there were no clinical manifestations. This is largely owing to the fact that the *x*-ray now shows much better detail of the colon than formerly, and probably the colon is being examined more routinely. We examined 615 cases of undoubted diverticula of the colon, but in only 137 were the symptoms sufficiently severe to warrant surgical intervention. We realize, however, that palliative measures can accomplish a great deal in some cases. The principal symptoms in our cases were pain, which was

present in nearly 80 per cent; constipation in about 60 per cent; abdominal tenderness in over 60 per cent; gas in the bowels in over 30 per cent; and palpable tumor in 34 per cent of the benign cases and in 31 per cent of those associated with carcinoma, thus showing that the presence of a tumor indicates diverticulitis rather than carcinoma, although this is not an important point in the differential diagnosis. When blood is present in the stool it suggests carcinoma rather than diverticulitis. Frequency and burning, and pain on urination often accompany diverticulitis of the sigmoid. Frequency was noted in more than 20 per cent of our cases. Cystoscopic examination may be of value in cases in which the colon communicates with the bladder. One of our patients, whose only symptom resulting from this condition was pus and mucus in the urine, had been treated a long time for cystitis, and the true condition was revealed only on cystoscopic examination. In the remaining 7 cases in which there was a fistula between the colon and the bladder, the diagnosis could be made readily from the presence of inflammation in the region of the sigmoid and the passage of gas and feces into the bladder.

Proctoscopic examinations are not of much value except to rule out other conditions. The lesion is likely to be too high to be reached with the proctoscope, and the deformity and angulation of the sigmoid make it inadvisable to attempt it. Fixation of the sigmoid may interfere with the passage of the sigmoidoscope in diverticulitis, and when found is corroborative evidence. We have seen the stoma of a single diverticulum through the sigmoidoscope, and also the rugous, fixed, mucous membrane lining a stricture due to diverticulitis.

In the differential diagnosis, if a mass is present in the sigmoid, the point of importance is to distinguish between diverticulitis and carcinoma. This is often impossible, and in many instances in which the abdomen is open and the tumor palpated it cannot be determined whether the lesion is diverticulitis or carcinoma, or diverticulitis and carcinoma. The history of repeated attacks of inflammation in the left lower abdomen

with subsidence of symptoms is the rule with diverticulitis. But the case in which the patient presents himself within a short time after his first attack, with a palpable mass in the bowel and partial obstruction still present, will always remain a diagnostic problem, so far as the differentiation of diverticulitis and carcinoma is concerned. The presence of blood or bloody mucus persistently in the stools indicates carcinoma. Ulceration of the mucosa in the strictured area of a diverticular involvement occurs occasionally, and severe bleeding may result; but this is rare. Persistent low-grade fever may be present with both conditions. Leukocytosis is a little more common with diverticulitis than with carcinoma, but there is not enough difference to be of value. We believe that, for the present at least, these two conditions must usually be distinguished by histologic examination of the tissues.

The *x*-ray reveals two types of diverticula: Those associated with spasm, inflammatory thickening of the intestinal wall and partial obstruction, seen most often in the sigmoid, and called diverticulitis, and those distributed in various parts of the colon, without spasm, thickening or narrowing, and called diverticulosis. When filled with barium the diverticula are seen as rounded or oval shadows projecting from the intestinal lumen. Such shadows, in conjunction with spasm or organic narrowing of the bowel, are pathognomonic of diverticulitis. The differential diagnosis is chiefly concerned with carcinoma, phleboliths, calcified glands, urinary calculi and barium pent up in contracted haustra.

Diverticula may fail to fill with barium if they contain fecal matter or have a stenotic inlet, and in such event diverticulitis may resemble carcinoma. Carcinomatous diverticulitis, if extralumenal shadows are present, cannot be distinguished from benign diverticulitis. By manipulation during the screen examination, shadows of the concretions mentioned may be shown to have no relation to the bowel. Normal barium-filled haustra are not accompanied by a filling defect and tend to disappear or to change their situation. Diverticular shadows maintain a fixed position and may persist after the bowel is emptied.

TREATMENT. In view of the fact that we have seen a number of these patients, even when they had an abscess or a tumor, entirely relieved by conservative treatment, we are tempted to follow this plan in a large percentage of cases. We are, however, constantly confronted with the fact that the condition may be malignant and that we are allowing an operable condition to become inoperable. We are convinced that the process is not progressive, and that unless there are symptoms from the diverticula no treatment other than palliative, such as regulation of the bowel movements, is indicated. Conservative measures rather than operation should be seriously considered in all of these cases. Operations have thus far been performed with a mortality of about 10 per cent. In operating, infection is the serious consideration. Although the tissues in these cases are not filled with bacteria to quite the extent that they are in cases of ulcerative colitis, nevertheless there is usually a virulent infection in the wall of the colon and mesocolon. While the patient may be combating this satisfactorily before operation, the manipulation of the tissues may result in spreading the infection rapidly. We have operated on a number of patients who had abscesses, either just draining the abscess or draining and suturing the opening left in the colon at the point of perforation of the diverticulum. In some instances the wound has healed readily, while in others fecal fistulas have persisted for a long time. If the condition is chronic and results in obstruction from tumor and angulation of the colon it is possible to resect the sigmoid and perform the anastomosis at one time, with satisfactory results in most instances. If, however, there is an appreciable amount of infection in or near the colon, or if there is a fistula from the colon leading into the bladder or other structures, the mortality will be reduced considerably by a preliminary colostomy, which accomplishes more than just the relief of obstruction. After the colostomy has been established the distal part of the colon, which is affected with the diverticulitis, can be flushed out thoroughly once or twice a day until all evidence of infection has disappeared; the resection can then be performed with comparative safety. Because of

the serious consequences following resection for diverticulitis, Mummery has advised permanent colostomy in some cases, being content with this procedure rather than resection at such risk. In most cases, however, we feel that after all the inflammation has subsided, resection and reëstablishment of the continuity of the colon can be attempted.

BENIGN DIVERTICULITIS

JANUARY 1, 1907 TO JANUARY 1, 1924

	Patients.	Per cent.	Hospital mortality.	Per cent.
Number	118	...	12	10.16
Males	84	71.18	8	9.52
Females	34	28.81	4	11.76

Oldest male	75 years	Oldest female	76 years
Youngest male	15 "	Youngest female	28 "

AGES BY DECADES

Years.	Patients.	Per cent.
11 to 20	1	0.84
21 to 30	1	0.84
31 to 40	12	10.16
41 to 50	37	31.35
51 to 60	42	35.59
61 to 70	18	15.25
71 to 80	7	5.93

BENIGN DIVERTICULITIS

JANUARY 1, 1907, TO JANUARY 1, 1924

	Patients.	Per cent.
Symptoms:		
Pain	90	76.27
Constipation	63	53.38
Abdominal tenderness	64	54.23
Gas in the bowels	42	35.59
Palpable tumor	41	34.74
Abdominal distention	27	22.88
Blood in stool	22	18.64
Painful bowel movement	20	16.92
Mucus in stool	15	12.71
Pus in stool	8	6.77
Liquid stool	3	2.54
Obstruction	11	9.32
Frequency of urination	24	20.33
Burning on urination	21	17.79
Pain on urination	17	14.40
Large amount of pus in the urine	11	9.32

BENIGN DIVERTICULITIS

JANUARY 1, 1907, TO JANUARY 1, 1924

FINDINGS AT OPERATION

	Patients.	Per cent.
Location of tumor:		
Sigmoid	88	74.57
Rectosigmoid	7	5.93
Colon	5	4.23
Cecum	5	4.23
Colon and sigmoid	4	3.38
Rectum	4	3.38
Not stated	5	4.23
Associated conditions:		
Adhesions	76	64.40
Perforation	31	26.27
Obstruction	25	21.18
Abscess localized in region of diverticula	14	11.86
Abscess in abdominal wall	3	2.54
Abscess in vesical wall	1	0.84
Abscess in rectal wall	1	0.84
Abscess in liver	1	0.84
Abscess under liver	1	0.84
Fistula into bladder	8	6.77
Fistula into sigmoid	1	0.84
Fecal fistula	1	0.84
Sinus of abdomen	1	0.84
Stones in the diverticula	4	3.38
Peritonitis	3	2.54
Associated findings:		
Gall stones	5	4.23
Large spleen	1	0.84
Carcinoma of the pancreas	1	0.84
Inoperable carcinoma of the rectum	1	0.84
Duodenal ulcer	1	0.84
Tuberculous glands and secondary infection	1	0.84
Cyst of the liver	1	0.84

BENIGN DIVERTICULITIS

JANUARY 1, 1907, TO JANUARY 1, 1924

	Patients.
Types of operation:	
Mikulicz	35
Tube resection	12
Resection	21
Colostomy	15
Excision of the diverticulum	15
Drainage of abscess	7
Anastomosis	7
Exploration	6
Total	118

Report of Cases

Case I (A433746).—*Extensive diverticulitis of the sigmoid relieved by cecostomy.* A woman, aged fifty-six years, had had abdominal exploration one month before coming to the Clinic, because of pain in the left lower quadrant, lasting one week. A tumor, 3.75 cm. in diameter, was found in the sigmoid, causing adherence to the broad ligament. A cecostomy was performed.

We found that the cecostomy was functioning satisfactorily and the patient felt well, but she desired to know the nature of the lesion. Extensive diverticulitis of the sigmoid was demonstrated by *x*-ray. With control of diet and irrigations the patient continued to improve, and finally was sent home without further operation. She reports that she has been entirely comfortable thus far.

Case II (A208146).—*Diverticulitis of the sigmoid without marked symptoms.* A woman, aged forty-six years, came to the Clinic because of intermittent and increased menstrual bleeding. She had had a goiter for twelve years and slight constipation.

Our examination revealed multiple uterine fibromas, and an adenomatous goiter which was not causing symptoms. At operation, September 21, 1917, multiple fibromas, causing a tumor about 12 cm. in diameter, were found in the uterus. At the rectosigmoidal juncture was a mass 3 cm. in diameter which showed evidence of perforation, as it was sealed against the peritoneum covering the posterior portion of the uterus near the internal os on the left side. A subtotal hysterectomy was performed and the rectosigmoid juncture resected. The pathologists reported that the segment of sigmoid contained diverticulitis.

The patient recovered uneventfully from the operation and returned home. When told of the presence of the diverticulitis the patient recalled that in the previous four years she had had occasional slight distress in the left abdomen accompanied by

constipation and inability to pass gas; she had been relieved by a bowel movement or flatus.

CASE III (A387471).—*Extensive diverticulitis of the sigmoid without marked symptoms.* A man, aged fifty-six years, had developed a sore mouth nine months before, and had noticed that he had a geographic tongue. Later his Wassermann reaction was reported to be positive and he was given treatment for syphilis. In June, 1923, he passed a small amount of blood with his stool. In the three months which have intervened his bowels have been regular until two weeks before coming to the Clinic, when he took Pluto water. This was followed at first by a bloody discharge and later by a severe hemorrhage, necessitating transfusion. Since the hemorrhage he had felt weak and sluggish, but had not had any pain or definite discomfort.

X-ray examination disclosed diverticula of the sigmoid, with partial obstruction. At operation, September 27, 1923, extensive diverticulitis and rather marked obstruction from long, thickened, adhesive bands of inflammatory diverticula were found. The involved area was brought to the outside as the first stage of the Mikulicz operation, and ten days later was cut off. The patient's convalescence was somewhat retarded on account of the marked secondary anemia, although two transfusions, of 500 cc each, had been given before the operation. The final stages of the operation were not completed, and after a month the patient was permitted to return home to complete his convalescence.

CASE IV (A387719).—*Perforating diverticulitis of the sigmoid, and vesicosigmoidal fistula.* A physician, aged sixty years, one month before examination, had had a sudden, sharp, lower abdominal pain, followed by fever, a mass in the left lower quadrant, frequency and burning on urination and the passage of gas by the urethra.

Examination revealed a great deal of pus in the stools, pus

2 and albumin 2 in the urine, hemoglobin 68 per cent and leukocytes 19,900. A median pelvic mass was palpable by rectum. Operation, April 24, 1922, revealed that the sigmoid and descending colon were thick and edematous; the descending colon was almost rope-like. The sigmoid was obstructed and densely adherent to the posterior wall of the bladder. All of the tissues were reddened, edematous and acutely inflamed; in some areas watery edema was present. The mesentery was thick and edematous. A left rectus colostomy was performed. About a year later inflammatory tissue around the colostomy wound and an anal fistulous tract were excised. No evidence of malignancy was demonstrable in the tissues.

In the two years since the primary operation the patient has regained his former weight, and he wrote recently that he was enjoying good health.

CASE V (A382782).—*Diverticulitis of the sigmoid causing obstruction and marked toxemia.* A woman, aged fifty-five years, during the last four years had had three attacks of extreme soreness in the left iliac fossa, accompanied by a rise in temperature and severe constipation. An abdominal exploration revealed a mass in the sigmoid, but no attempt was made to remove it.

The nature of the tumor was of the greatest concern to the patient, although marked obstruction was evident at the time of our examination. The *x*-ray revealed multiple diverticula of the sigmoid and descending colon. The hemoglobin was 67 per cent and the leukocytes 9900. At operation, February 4, 1922, diverticulitis of the sigmoid had caused a suspicious-appearing tumor. The Mikulicz operation was performed in four stages. The pathologists reported multiple diverticula, averaging 1 by 0.5 cm. in diameter, and peridiverticulitis. Microscopically there was no evidence of malignancy. About 30 cm. of bowel were removed.

The patient left our care in April, 1922, in good condition, and when last heard from was improving steadily.

CASE VI (A93664).—*Diverticulitis of the sigmoid with obstruction and spontaneous abdominal and vesicosigmoidal fistulas.* A man, aged forty years, began to have abdominal cramps five years before coming to the Clinic. In an attack four years before he had had chills, fever and pain in the left lower quadrant, followed by superficial inflammation. The inflamed area was incised and drained, but there was a rather persistent discharge of pus. One year later it was necessary to repeat the procedure, and several abscess pockets were evacuated. A third attack occurred one year before the visit to the Clinic, and was followed by a discharge of pus through the old sinus tract and also through the urethra. Two operations did not relieve this condition.

A fecal fistula was found in the left groin; it had been opening and closing intermittently for a year. The *x*-ray examination of the colon was unsatisfactory on account of the loss of bismuth through the fistula. Operation, October 22, 1918, disclosed dense adhesions throughout the pelvis. A ruptured diverticulum of the sigmoid was discharging through the fistulous tract. The intestinal loop above the area of diverticulitis was dilated to three times normal. The involved sigmoid was resected and anastomosed by a tube.

The patient was dismissed from observation on the thirtieth day after operation in good condition, and has not been heard from since.

CASE VII (A453119).—*Perforating diverticulitis of the sigmoid and vesicosigmoidal fistula.* A man, aged fifty years, had had several attacks of constipation or diarrhea, associated with abdominal cramps, fever and, at times, chills for eight years. For the past ten days repeated cathartics had failed to obtain a bowel movement, although enemas resulted in the passage of a little gas and a small quantity of fecal matter.

Examination revealed marked tenderness over the descending colon, normal temperature and 12,000 leukocytes. *X*-ray examination of the colon disclosed a filling defect in the sigmoid and obstruction suggestive of diverticulitis. A day or two later

the patient suddenly developed frequency of urination, dysuria, and passed gas by urethra. There was increasing toxemia and distention of the abdomen, especially the left lower quadrant. With a dietary régime and laxatives the toxemia decreased, and the bowels moved freely during the following two weeks. Operation, February 11, 1924, disclosed acute perforating diverticulitis, communicating with the bladder. Colostomy was performed and no further exploring done on account of the acuteness of the condition.

Recovery was uneventful, and the patient left our care on the twenty-first day, feeling well.

Case VIII (A442388).—*Diverticulitis of the sigmoid. Relief of symptoms by medical management.* A man, aged sixty years, had had mild lower abdominal distress, easily relieved by laxatives for one year. One month before he had had an attack of severe lower abdominal pain followed by a rise in temperature, tenderness and rigidity in the lower left quadrant, and had been kept in bed for three weeks, because of swelling of the abdomen; this subsided.

Our examination disclosed nothing grossly abnormal. *X*-ray examination of the sigmoid demonstrated the presence of diverticula. Mineral oil and a restricted diet were advised, and the patient left for home, October 1, 1923.

In a report dated February 1, 1924, he states that he has gained 20 pounds, is not entirely free from symptoms, but has had only one day of severe discomfort, and that occurred within the last month.

Case IX (A376337).—*Diverticulitis of the sigmoid and emphysema of the scrotum.* A physician, aged forty-six years, in the last three years had had two attacks of intermittent left lower abdominal pain, lasting eight and six months, respectively. The pain increased with defecation; it was accompanied by constipation and at times a rise in temperature. There was frequency and pain and burning on urination.

Urinalysis revealed pus 3 and albumin 1 in the urine. A rather marked cystitis was present. Rectal palpation revealed a mass in the region of the sigmoid, and the *x*-ray the presence of diverticulitis, causing obstruction. At operation, November 9, 1921, diverticulitis of the sigmoid was found. A loop of the small intestine (the ileum) had become attached to the mass and the posterior wall of the bladder, due to the inflammatory reaction, and the descending colon was dilated, giving evidence of obstruction. The infection was acute at the time, and there was considerable edema and inflammation around the tumor. Primary resection seemed inadvisable, because so many structures seemed to be involved, and a left rectus colostomy was made. November 6, 1923, after a long tedious dissection, the sigmoid was freed and resected and an end-to-end anastomosis made.

On the seventh day after the colostomy the patient rapidly developed an emphysema of the scrotum, but after puncture of the colostomy loop the condition subsided. In the two-year interval between the operations the patient gained 50 pounds. He is now waiting to have the colostomy closed.

Case X (A448543).—*Perforating adenocarcinoma and diverticulitis of the sigmoid; carcinoma of the ileum and obstruction.* A man, aged fifty-eight years, had had three or four attacks of abdominal cramps eight years before, which were said to be due to appendicitis. Six months before, constipation had increased, and for three months was accompanied by abdominal cramps. Six weeks later he noticed a frequent desire to got to stool; the feces were liquid and contained much mucus. From this time on he lost strength and 25 pounds in weight. A few days before his visit to the Clinic he vomited for the first time.

Examination revealed tenderness over the lower abdomen, blood and excess mucus in the stools and obstruction in the sigmoid. About forty-eight hours after examination of the colon the patient developed tenderness and severe pain in the lower left abdomen, and became toxic. A definite mass was palpated at the level of the pelvic brim, but this disappeared after three

weeks of hospitalization. Proctoscopy at this stage revealed a fixed lesion 20 cm. beyond the sphincter. On mineral oil and a soft diet he improved a great deal during the next three weeks. Operation, January 18, 1924, disclosed an adherent sigmoidal mass at the brim of the pelvis, which gave the feel and appearance of a carcinoma. The sigmoid was indurated above the mass. There was no evidence of distant metastasis. A colostomy was performed, and a month later the mass, which consisted of sigmoid, a loop of adherent ileum and the appendix, was removed. There was an annular adenocarcinoma of the sigmoid, 4 cm. long, and a carcinoma, 2 by 1.5 cm. long, in the ileum. Glandular involvement was not demonstrable. Small diverticula were found in the bowel both above and below the growth. March 25 the colostomy was closed and the continuity of the bowel reëstablished. The patient's convalescence has been uneventful.

Case XI (A147966).—*Perforating diverticulitis of the sigmoid and mesenteric abscess.* A man, aged fifty-two years, had had attacks of cramping pain in the left lower abdomen for one year. Enemas and mineral oil afforded moderate relief. The left lower abdomen had been tender since an attack of abdominal pain three weeks before. The bladder was irritable at this time.

Examination revealed tenderness and a mass in the left lower quadrant. *X*-ray examination of the sigmoid revealed diverticulitis. Operation, December 28, 1915, disclosed a tumor of the sigmoid, 10 cm. long, closely adherent to the pelvic wall, extensive inflammatory reaction in the surrounding tissues and an abscess in the mesentery, with the tip of the appendix drawn into it. The appendix was removed and the first stage of the Mikulicz operation performed, bringing the tumor and 25 cm. of the bowel to the outside through an incision in the left rectus muscle. January 5, 1916, the second stage of the Mikulicz operation was performed and the tumor removed. January 18 the third stage was performed. The patient recovered satisfactorily.

CASE XII (A399677).—*Diverticulitis of the sigmoid and vesico-sigmoidal fistula.* A man, aged sixty years, had had attacks of pyrexia for five years, and had noticed large amounts of sediment in the urine, and for three years flatus at the end of micturition.

Urinalysis revealed albumin 2 and pus 3. Cystoscopic examination revealed an opening in the wall of the bladder through which fecal matter could be seen. *X*-ray examination of the colon revealed diverticulitis. At operation, August 29, 1922, it was found that the opening in the bladder had occurred at about the middle of the left lateral wall. Many diverticula were present in the sigmoid above the point of its attachment to the bladder. The sigmoid and bladder were separated and the openings closed.

The convalescence was uneventful, although prolonged on account of an indolent superficial fistula in the region of the left groin; this eventually healed spontaneously.

CONCLUSIONS. 1. Diverticulitis of the colon is more common than was formerly believed. There may be a solitary diverticulum in any portion of the colon, or a great many diverticula distributed from the ileocecal valve to the rectum.

2. Diverticulitis is apparently not progressive and may remain unchanged for years. In many instances it apparently does not cause symptoms.

3. Diverticulitis is a disease of middle life. The condition we speak of as diverticulitis is peculiar to the sigmoid. We have not observed the condition, which is a diffuse edema, inflammation and swelling throughout all of the tissues of the colon and mesentery, in any other quadrant of the colon.

4. As the inflammation in the diverticula and wall of the colon progresses, an abscess may form and later perforate into the abdominal wall, bladder or intestine. If the inflammation remains chronic a tumor, composed of fibrous tissue and the products of inflammation, develops.

5. It is often difficult to distinguish between diverticulitis of

the sigmoid and carcinoma; in a number of the cases the conditions are associated. Just what influence diverticulitis has on the development of carcinoma we are unable to determine. If the history reveals that the patient has had repeated similar attacks and has noticed a tumor for a long time, increasing and receding from time to time, the probability is that it is due to a diverticulitis. In some instances a definite diagnosis cannot be made until the tissues have been examined histologically.

6. Many patients with diverticulitis can be relieved by dietary and medical management.

7. If an abscess forms from the perforation of a diverticulum or extension of the infection through the wall of the colon, drainage is indicated. If perforation occurs into other regions an operation is the only procedure that offers relief.

8. If there is any question as to the nature of the tumor operation is indicated.

9. The mortality from radical operations for diverticulitis has been very high. The difficulty in these cases has arisen from stirring up the infection that existed in the tissues around the colon before the operation. It seems to us, from a review of the results in these cases, that the plan of procedure should be a preliminary colostomy for the purpose of caring for any obstruction in the colon, and particularly the reduction of the inflammation in the diverticula by frequent irrigations of the lower colon. Resection of the infected portion can then be made with less risk.

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MULTIPLE PRIMARY MALIGNANT FOCI IN CANCER OF THE COLON

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During a recent study of cancer of the colon carried on in the laboratory of surgical pathology in the Johns Hopkins Hospital, which was reported last spring to this Association, there were encountered certain cases of multiple cancers of the intestine. Two of these, with possibly a third, appear to be instances of the development of two or more primary cancers in one individual; 2 other cases are probably examples of recurrence, but with so great a period of time elapsing between the first and second tumors as to introduce an element of uncertainty. The subject has proved of sufficient interest to warrant a brief communication, which is made, be it said, with full realization of the extreme difficulty, often the utter impossibility, of determining convincingly the exact status of any case in this category. In view of the general and well-founded clinical conception of cancer as a condition arising from a single focus of origin, but acquiring multiple secondary foci of development, the burden of proof is indeed upon one who assumes a case to illustrate multiplicity of primary malignant tumors, and there are so many possibilities of error in observation and interpretation that one necessarily approaches this subject with extreme reserve.

Five of the 129 cases previously studied[1] form the basis of this report:

S. N., 16588. A white man, aged forty-nine years, was admitted to the Johns Hopkins Hospital, having had for six

weeks intermittent colicky pain, and for four weeks a palpable abdominal mass. He had lost forty pounds in weight. Upon exploration there was found an inoperable carcinoma of the cecum, and an anastomosis was made between the ileum and transverse colon. Symptoms of obstruction persisting, a second

FIG. 1.—S. N. 16588. Photomicrograph of cancer of ileum.

exploration was made four days later and revealed a stenosing carcinoma of the sigmoid. Colostomy was without avail and the patient died following this operation. At autopsy there were found: (1) A large cancer completely encircling the ileum and extending into the lumen so as to produce marked obstruction; (2) a second similar tumor at the cecum involving the

ileocecal valve and extending around the lumen of the large intestine (this growth was surrounded by a number of polypoid masses and extended deeply into the mesentery); (3) a third tumor, evidently cancerous, surrounding the bowel at the sigmoid flexure. Microscopical examination showed the tumors of the

FIG. 2.—S. N. 16588. Photomicrograph of cancer of cecum.

ileum and of the cecum to be adenocarcinomata of similar histological appearance, both showing a pronounced tendency to invade. The muscular coats had been broken through and the subperitoneal fat invaded; a small lymph gland lying in the mesentery just below one of the tumors was involved, but otherwise the lymph glands were negative. The growth of the sigmoid

flexure was rather more papillomatous in type, there being practically no invasion of the muscle. The glandular arrangement was preserved throughout this tumor, in some places the alveoli being lined by a single layer of cells and in other places by multiple layers of cells. This case was discussed briefly in 1904 by Bunting,[2] who remarked that "the author leans toward the view in this case that the foci are independent primary foci." (Figs. 1 and 2.)

FIG. 3.—S. P. N. 14328. Cancer-bearing sector of colon which was removed at second operation.

S. P. N., 14328. A white man, aged forty-three years, presented a history of constipation for many years, for one year a definite change in the stools and a loss of 20 pounds in weight. Examination disclosed a carcinoma of the rectum 12 cm. from the anal margin. This growth was removed and proved to be a typical adenocarcinoma, with infiltration through the wall of the bowel toward the fat in the hollow of the sacrum. No involved lymph glands were found. Eight years later the patient suffered an acute intestinal obstruction, and at operation there were found two distinct carcinomata, one in the hepatic flexure

FIG. 4.—S. P. N. 14328. Low power photomicrograph of the adenocarcinoma. A small portion of normal mucosa is seen at the right end of the section.

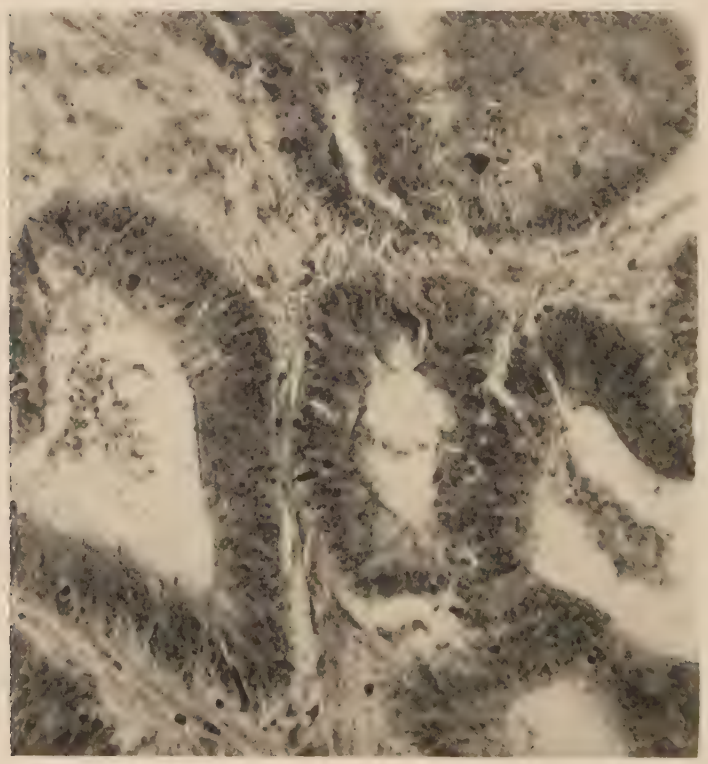

FIG. 5.—S. P. N. 14328. High power photomicrograph of the adenocarcinoma showing the preservation of the glandular arrangement.

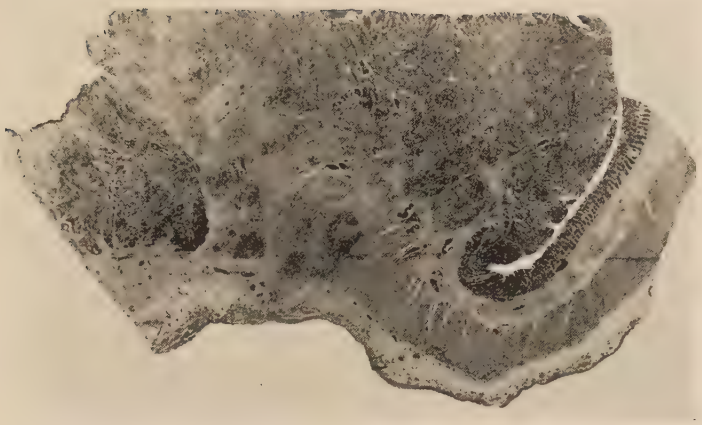

FIG. 6.—S. P. N. 14328. Low power photomicrograph of the smaller more solid tumor. Normal mucosa is visible towards the right end of the section. Note the striking differences between this tumor and its companion tumor, Fig. 4.

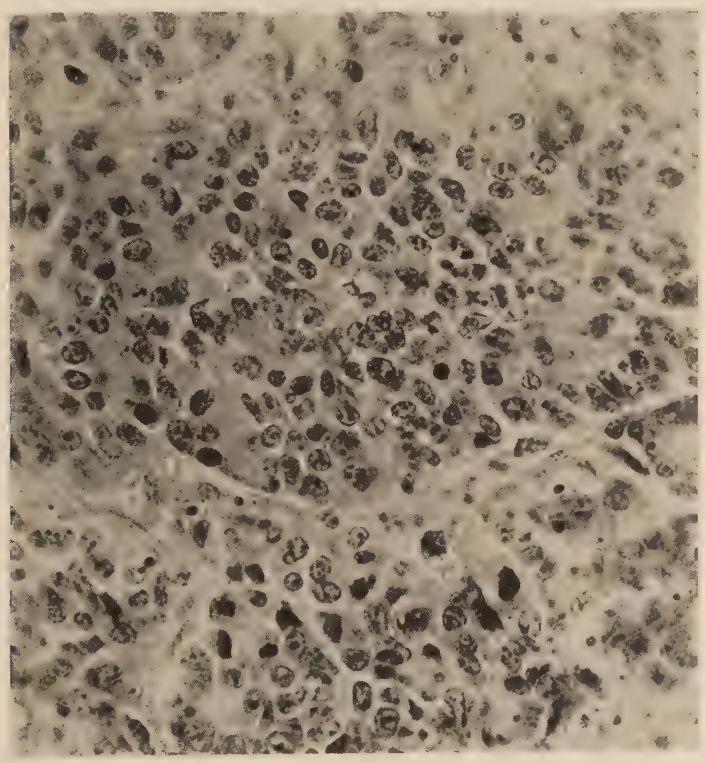

FIG. 7.—S. P. N. 14328. High power photomicrograph of tumor shown in Fig. 6. Note the great difference between this section and Fig. 5, its companion tumor.

and the other in the right part of the transverse colon. That portion of the intestine bearing the growths was successfully resected, the patient dying three months later from an unrelated cause. Pathological examination showed one of this pair of cancers to be typical adenocarcinoma, with a marked tendency to form glandular acini, while the other presented a marked histological resemblance to squamous-celled cancer, though here and there preserving a slight but definite tendency toward an alveolar arrangement. There were metastases to the lymph glands at the hepatic flexure. (Figs. 3, 4, 5, 6, and 7.)

FIG. 8.—S. P. N. 15059. Longitudinal section through the sigmoid loop and its mesentery. The mucosa is shown above and the mesentery below. Two cancers present in the lumen of the bowel, that at *A* being the larger tumor and preserving its adenocarcinomatous structure, that at *B* being a smaller and more fibrous tumor.

S. P. N., 15059. A white man, aged seventy-two years, on the fourth day of an acute obstruction submitted to cecostomy, dying thirty-six hours later. At autopsy there were found two annular carcinomata, one at either foot-point of the sigmoid.

The upper tumor, which was confined to the wall of the bowel, presents so characteristic a histological picture of adenocarcinoma that it could well typify the large majority of cancers of the colon; the lower tumor, which had invaded the mesentery and become adherent to the bladder, presents quite a different picture. It has invaded the muscular coats deeply, and the epithelial cells are throughout closely packed together with very little tendency to take a glandular arrangement. Here and there

FIG. 9.—S. P. N. 15059. Low power photomicrograph of tumor *A*.

one recognizes a group of cells with a somewhat alveolar arrangement, but in the main this has been lost, even in the advancing edge of the tumor which one sees just beneath the normal mucosa. The lower tumor is apparently somewhat older than its fellow, which is still contained by the muscular coats of the bowel. There are quite marked differences in the histological appearance of the two tumors, and these differences are not readily explained as representing different stages of growth in identical tumors.

FIG. 10.—S. P. N. 15059. High power photomicrograph of tumor *A*. Note the preservation of the glandular arrangement.

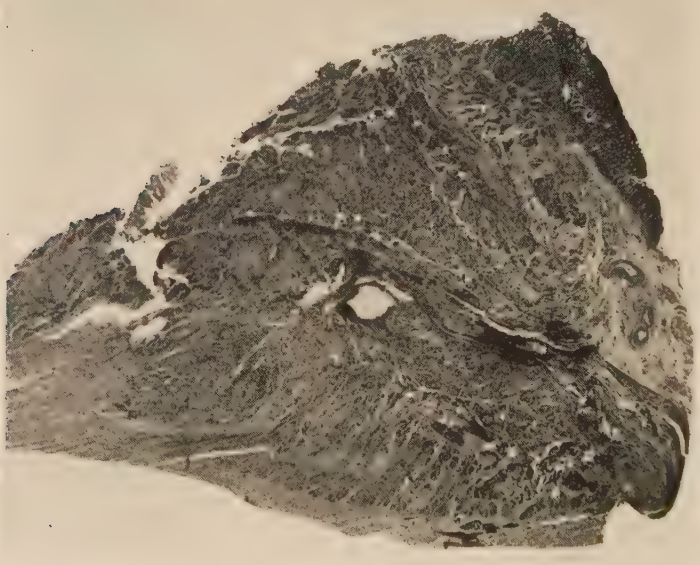

FIG. 11.—S. P. N. 15059. Low power photomicrograph of tumor *B*.

Both tumors have evidently started in the mucosa; neither represents secondary involvement of the mucosa by extension from a subperitoneal or mesenteric metastasis of the other tumor. These three points are important evidence bearing directly upon the question of multiple primary cancers. (Figs. 8, 9, 10, 11, and 12.)

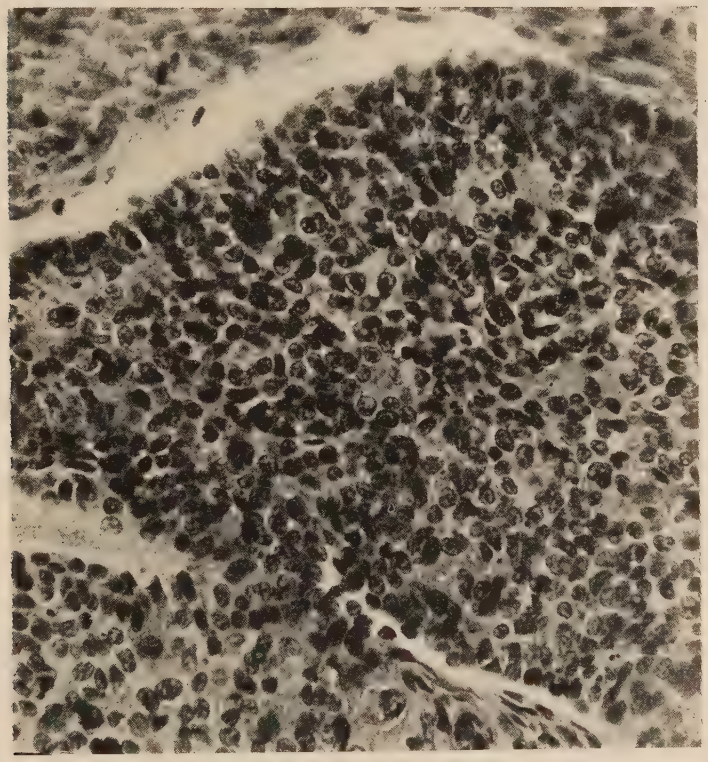

FIG. 12.—S. P. N. 15059. High power photomicrograph of tumor *B*. Note the closely packed epithelial cells, strongly suggesting squamous cell cancer. There is a striking difference in appearance between this tumor and that shown in Fig. 10.

S. P. N., 15001. A white man, aged sixty-two years, on the seventh day of an acute intestinal obstruction submitted to cecostomy. Three months later a cancer of the sigmoid was

FIG. 13.—S. P. N. 15001. Cancer of the sigmoid.

FIG. 14.—S. P. N. 15001. Metastasis to adjoining lymphatic glands.

resected, and on examination was found to be a rather fibrous type of adenocarcinoma, producing stricture of the gut. The lymphatic glands were invaded. The patient died seven years later, with the clinical signs of cancer of the cecum involving the abdominal wall at the site of the cecostomy. Unfortunately no autopsy was made. (Figs. 13 and 14.)

S. P. N., 6595. A white woman, aged thirty-eight years, submitted to exploration because of suspicious symptoms; there was found a carcinoma of the cecum, which was successfully resected. Sixteen years later the patient died; a diagnosis of cancer of the liver, though not verified by autopsy, seemed quite secure.

These 5 cases represent a variety of conditions, but there is common to them all the fact of multiple cancers, which in each instance occur in such fashion as to throw open to reasonable doubt the assumption of a single primary tumor as the sole point of origin of the disease. Such doubt may be aroused by the presence in the intestine of several cancerous foci, among which there is great similarity in size, extent of invasion and apparent age. A similar suspicion is aroused by the occurrence of two or more cancers widely separated by an intervening length of healthy gut, particularly when the aboral tumor appears to be the oldest of the group; if several years intervene between the various tumors and the aboral member of the group is unmistakably the oldest, as in the second instance presented above, the conclusion as to multiplicity can scarcely be avoided. Marked histological differences between the various tumors are difficult to explain other than by their independent origins. The question of recurrence or a second independent tumor is usually open and has provoked much argument; under certain conditions, as marked delay in the appearance of the second tumor, we cannot hope to arrive at entirely definite conclusions, since either is a possibility and we lack reliable criteria. Thus the question of multiplicity is raised by each of the 5 cases, and its discussion, even though inconclusive, is not entirely devoid of interest.

The regularity with which cancerous disease takes its origin from a single parent tumor has been so borne in upon us that the occurrence of a case suggesting multiplicity of origin excites a definite interest. The condition has been recognized for many years. Billroth is commonly credited with the first case report, which appeared in his *Manual of General Surgical Pathology* in 1860, and told of a cancer of the stomach found in a man from whom an epithelioma of the external ear had been previously removed. Mercanton[3] states that Rokitansky, in 1855, and von Bruns, in 1859, both mentioned the condition, and that between 1863 and 1889 the subject was considered by a number of men, among whom are Virchow, Volkmann, Tillmann and Schimmelbusch. The early reports usually considered multiple cancers of the skin, and it was more or less through the development of this theme that attention was directed toward the other forms of multiplicity. It is probable that multiple primary carcinomatous foci of the skin are associated in the minds of most clinicians with the dry, senile type of eczema and with *x*-ray dermatitis; but a number of other conditions are mentioned in the same connection, as paraffin and tar cancer, chimney-sweepers' cancer of Percivall Pott, arsenic cancer (in which a period of pachydermia precedes the development of malignant foci) and the epitheliomata which develop in tissue changed by lupus or psoriasis. Von Hansemann[4] emphasizes his belief that only those forms of skin cancer are regularly multiple which are preceded by an inflammatory affection of the skin. With increasing knowledge it became evident that the many examples found in the skin could be conveniently considered together, thus forming one of the clinical groups rather generally adopted at present, viz., primary multiplicity affecting a single organ, the skin and the gastrointestinal tract being typical examples. Besides this group, there are today usually recognized two others, viz., primary multiplicity affecting paired organs and primary multiplicity in which the sites of the several tumors are unrelated. There are many examples of each of these groups.

When two or more carcinomata appear simultaneously, it

may be less difficult to determine their independence of each other than when these tumors appear at intervals of several years, since in the second instance one must distinguish between recurrence and new growth. Distinct histological differences between the two tumors are usually accepted as establishing independence, but it does not necessarily follow that histological similarity means one parent tumor from which the others have sprung; multiple independent carcinomata of the colon developing on a groundwork of polyposis serve well as an illustration. A few years ago a patient sought examination because of a tiny "lump" in the right breast. The suspicious area was so very small that diagnosis other than by microscopical study could not be made, and since there seemed little or no chance of the mass being malignant, it was excised under local anesthesia. The mass proved to be carcinomatous, and radical operation was promptly made. Careful study of the breast in the laboratory revealed a second malignant focus, smaller even than the first, from which it was separated by a wide area of normal breast. The patient has remained well. Such a case is surely an instance of simultaneous origin of multiple primary malignant foci. For the purpose of argument, however, let us suppose that with the little mass removed nothing more had been done until the second independent cancer, left undisturbed, had grown large enough to be recognized as carcinoma of the breast; under these conditions a clinical diagnosis of recurrent cancer would have been accepted without question. Ellsworth Eliot[5] has discussed this subject recently and presented a number of interesting cases. Ewing,[6] in discussing the influence exerted by tumors upon surrounding tissues as a source of recurrence of the growth, remarks that this question is closely related "to that of the multiple origin of tumors in the same organ."

In the study of this condition, therefore, one must consider the time as well as the site of occurrence. Theilhaber[7] states that in point of frequency of occurrence the condition of multiple primary carcinomata takes the following order, viz: (1) Local disease which affects one system only, as the gastrointestinal

tract, and usually but a part of that, as the colon; (2) disease of similar paired organs; (3) synchronous, widely separated foci of disease; and (4) widely separated foci of disease appearing at reasonably long intervals.

Early in the study of these cases Billroth formulated three conditions which he thought must be fulfilled by multiple cancers before the different tumors can be considered independent of each other, namely, (1) the two growths must show distinct histological differences, and these must be so pronounced as to exclude their interpretation as merely different stages of development; (2) each growth must spring from its parent epithelium; (3) each growth must have its own group of metastases. Mercanton attributes these criteria to Michelsohn and Küster, but he apparently is alone in failing to credit them to Billroth. It is obvious that simultaneous tumors which fulfil each of these three conditions would have very strong evidence of their independence of each other. Mercanton adds a fourth condition to the effect that if after the removal at one operation of two cancers the patient remains free from disease, it is practically certain that the two growths were independent, since had either been a metastasis it would be entirely reasonable to assume the presence of other metastases, a state of affairs incompatible with life. This fourth condition appears sound, and is applicable particularly to multiple cancer of the colon, and similar examples where the growths are confined to one relatively small organ. There are apparently unquestioned instances of multiple primary cancers of the colon, and it is evident that such a case may not fulfil Billroth's three conditions. Bunting is of the opinion that these criteria were obviously intended to apply to carcinomata arising in different organs. Surely, any group of simultaneous lesions which fulfil all of these conditions would be beyond doubt. Indeed, it seems quite safe to accept certain cases which fail to meet all of them. Arbitrary insistence is unreasonable; commonsense recognizes, probably without argument, the primary multiplicity of adenocarcinomata of the colon which start in polyps. There is much in the literature indicating that a colon which is

the site of multiple polyps is prone to present cancerous changes in certain of them, so that whether these tumors appear simultaneously or, as in our second case, spaced by several years, one can hardly doubt the nature of the condition. Fig. 15 shows this not infrequent finding, viz., cancer and polyp in close association. (Figs. 15, 16 and 17.)

FIG. 15.—S. P. N. 27377. The cecum has been opened to show the mucous surface. There is seen a large adenocarcinoma at whose edge (to the right) is situated a benign polyp.

In the gastrointestinal tract, however, there are a number of ways in which a secondary dependent growth may become established, so that our problem is often very difficult. We recognize metastasis by the lymphatics; commonly we think of this as occurring by a path leading directly and consistently away from the tumor, but there is always the possibility of metastasis by retrograde or other circuitous routes. It may be a matter of the utmost difficulty, indeed impossible, to assert concerning two cancers of the gastrointestinal tract that each is a primary neoplastic unit quite independent of its companion tumor. Our case of double cancer of the sigmoid presents just

this problem: This patient was brought to the hospital in acute obstruction of the sudden unheralded type. Colostomy failed to rescue the patient, and at autopsy there were found two cancers, one at either foot-point of the sigmoid. One was confined to the wall of the bowel, the other had begun to penetrate it. The growths were not in contact nor were there any visible

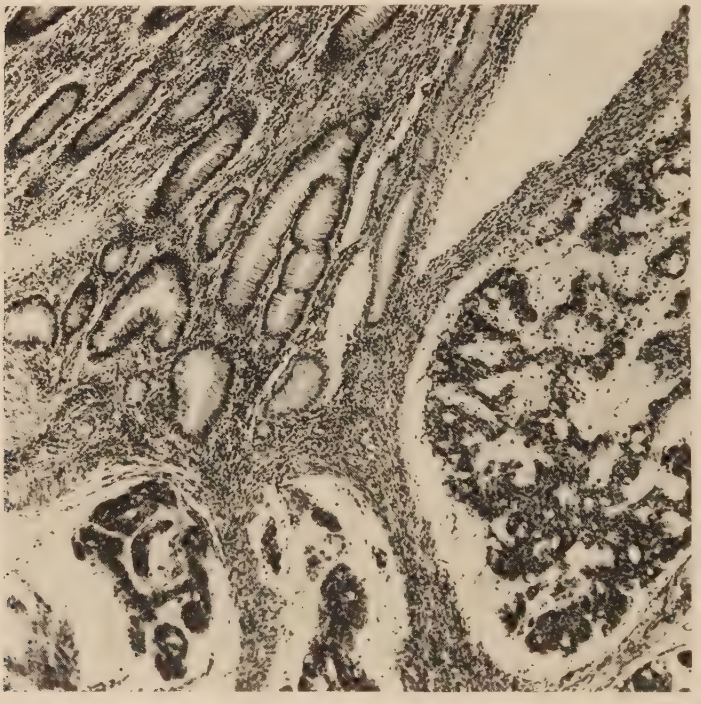

FIG. 16.—S. P. N. 27377. Photomicrograph of adenocarcinoma in Fig. 15.

lymphatic strands connecting them; they were approximately of the same size, from which one may infer their approximately equal length of life. Under these circumstances there exists at least the possibility of their simultaneous development. It is similarly possible, however, that lymphatic metastasis has occurred by way of the mesentery, traveling first upward to its root, and then, perhaps because continued progress was

blocked in this direction, down to the other foot-point of the sigmoid. Had this occurred early in the disease the final picture might simulate double primary cancer closely. Were a case such as this to fulfil the three conditions of Billroth, one would be inclined to accept the primary multiplicity of the carcino-

FIG. 17.—S. P. N. 27377. Photomicrograph of benign polyp in Fig. 15.

mata as proved; it must be recognized, however, that failure to meet the conditions does not necessarily throw the case out. The possibility of the occurrence of primary multiple carcinomata which do not fulfil Billroth's conditions cannot be denied.

We recognize the occasional transmission of cancer also by

the blood stream, it even being held that retrograde venous embolism may occur. There is also the establishment of a second cancerous focus through contact; certain cases in the literature illustrate the transmission of cancer from one to the other lip or from the tongue to the cheek. In the gastrointestinal tract this condition usually brings about the adherence of one organ to the other, the final appearance being that of direct extension of cancer in continuity. Dissemination through the escape of cancerous cells which float off in the peritoneal fluid is well recognized. Such secondary growths are frequently found in the pelvic parietal peritoneum, and their occurrence is not infrequent upon the visceral peritoneum, particularly of the omentum and the mesentery. We assume this to be a late phenomenon resulting usually in a multitude of small, obviously secondary growths, so that simulation of but two or three primary carcinomata could hardly result.

There is an implantation type of cancer in the gastrointestinal tract which is of very considerable interest. Indeed, this mode of transmission is usually held to account satisfactorily for multiple carcinomata presenting in the lumen, and is so obvious that it is difficult to avoid its acceptance when the growths are of similar histological type, and so situated as to indicate that the aboral member is the youngest of the group. However, when a prolonged period of time separates the appearance of the two tumors and the second lies orally with reference to the first, as is illustrated in the second of the cases cited above, implantation or engrafted cancer fails to explain the situation satisfactorily. A striking example of implantation cancer of the gastrointestinal tract is reported by L. Hoche[8] in *La Presse médicale* of 1901, and is of sufficient interest to warrant its statement in detail:

A man, aged seventy-five years, died from cachexia twelve hours after admission to the hospital. At autopsy there was found an epithelioma of the esophagus, 4 x 2.5 cm., situated 5 cm. above the bifurcation of the trachea. The lesser curvature

of the stomach was adherent to the pancreas, and upon opening the stomach there was found in this situation a chronic ulcer the size of a 2-franc piece. On the base of the ulcer was a hard nodule which was not in contact with the epithelial edge of the ulcer at any point, and which, though more or less embedded in the pancreas, was quite distinct from it. Histologically the nodule proved to be a discrete, sharply outlined cancerous mass

FIG. 18.—Section taken on the edge of the ulcerated tumor of the esophagus. (Reproduced from article by Hoche.)

which did not invade the pancreas. The base of the ulcer was otherwise composed of granulation tissue, and its epithelial edges showed nothing more than simple inflammation. The pancreas likewise showed only simple inflammatory changes. The lesion was apparently a simple ulcer in whose center there had developed a cancer which entirely lacked contact with normal epithelium at any point. The growth in the esophagus

(Fig. 18*) was found to be a typical squamous epithelioma whose histological picture was duplicated by the carcinomatous nodule in the base of the ulcer (Fig. 19*). Nothing was found to suggest the usual adenocarcinoma of the stomach. No other cancerous lesions were found.

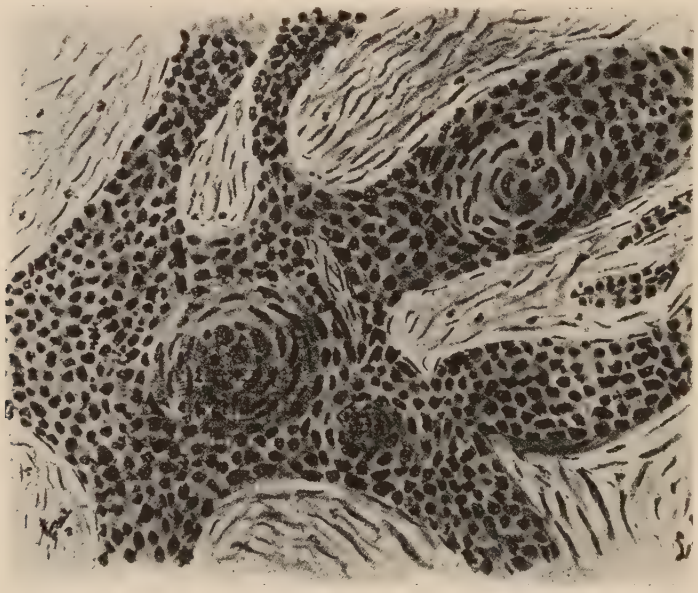

FIG. 19.—Section taken in the neighborhood of the base of the gastric ulcer (Reproduced from article by Hoche.)

Another striking example of engrafting of cancer was reported by Chalier[9] in 1909: A man, aged forty-three years, came to autopsy some months after an anastomosis between the ileum and the transverse colon had been made to relieve him of partial obstruction due to a cancer of the cecum. There were found (1) in the ileum, 10 cm. from its termination, three or four submucous nodules as large as a small chestnut, (2) in the cecum an ulcerating cancer of the ileocecal valve, (3) a stenosing cancer at the junction of the cecum and the ascending colon, and (4)

* Reproduced from article by Hoche in La Presse médicale, 1901, i, 67.

in the transverse colon thick, sclerotic cancerous tissue surrounding the anastomosis. Histologically the growths were all colloid cancer. Chalier remarks that the growth in the transverse colon is definitely an implanted cancer, since it occurs in tissues which were quite healthy at the time of operation, but which suffered both operative trauma and the scarring incident to healing. One can well imagine that implantation occurred upon some granulating area before the healing process had been completed. Dowden,[10] in 1917, reported a case of much the same sort. The patient was a woman, aged sixty years, who submitted to three operations: (1) Resection of a cancer of the sigmoid, followed in three years by (2) colostomy for cancer of the rectum, and at the end of a year (3) resection of a cancer of the small bowel. A few months after the final operation cancer appeared at the colostomy wound. Dowden believed the disease to have been primary in the ileum. Recurrence or the development of a second cancer in the traumatized tissue of an old colostomy is shown in our Case IV; here, however, there can hardly have been a question of implantation, since the first tumor was in the sigmoid and the second in the cecum. Fenger,[11] in 1888, reported a case which may be interpreted as double primary cancer of the colon or as an example of implantation. It concerns a man, aged about forty-five years, upon whom an anastomosis between the ileum and the transverse colon was made for cancer of the ascending colon. At autopsy, made ten days later, there was found a cancer of the ascending colon, producing complete obstruction, and a second cancer of the splenic flexure, causing marked stenosis.

There appears to be no doubt as to the occurrence of implantation cancer of the mucous surface of the gut, and of all the forms of secondary growth, apparently cancer so produced may most clearly simulate primary cancer. This is particularly true when the growths are limited to a small section of the gut, as, for instance, the colon. Practically all cancers of the colon, whether primary or secondary, are assumed to preserve an adenocarcinomatous structure in a more or less marked form, so that a widely

different histological appearance in the two growths is not expected. They all spring from epithelium of the same type—the mucosa of the large gut—and in general all would tend to produce metastases of the same type, though in different situations.

A much more fundamental question, indeed, has been raised from time to time as to whether the lesion diagnosed clinically as cancer has in fact started in and grown from one center, or whether its unity does not rather mean that since the process started, enough time has elapsed to allow coalescence by growth of multiple centers of origin, thus converting actual multicentric growth into apparent unicentric growth. This carries the question beyond the limits of clinical study; diagnoses from physical signs cannot be made in such early stages of disease.

The foregoing discussion makes no pretense of attempting more than a brief *résumé* of the subject, together with the presentation of several cases which illustrate certain aspects of the problems of the genesis and the growth of cancer. Although the matter appears to be largely of speculative interest, its consideration, nevertheless, brings out certain facts of some importance in the management of cancer of the colon. It must be recognized that there occur certain rare cases in which cancer of the colon starts in two or more primary foci. The terminal ileum is not uncommonly associated with the colon in this type of disease. Exploration of the ileum and entire colon should therefore be a routine step in the operative treatment; experienced surgeons have closed the abdomen without suspecting the presence of the second tumor. A growth of the splenic flexure is peculiarly elusive.

By a process of implantation secondary cancers may be engrafted on the mucous surface of the colon or elsewhere in the gastrointestinal tract, and it appears that traumatized tissue is an especially fertile bed for this development; such an occurrence is not rare and may result in a condition offering the same surgical problems as multiple primary tumors. Adenocarcinoma, the common type of cancer of the colon, seems especially apt to develop secondary implantation growths.

Clinical experience and many reports in the literature make it plain that carcinoma is prone to develop in the mucosa of a polyp of the colon. There are numerous records of the association of polyposis and cancer; there may be found a cancer and a polyp side by side, as pictured above, or in a colon bearing numerous polyps there may be found two or more cancers apparently of approximately the same age. In the literature there are reports of cases, like our second case, in which two or more cancers develop one after the other and separated by intervals of several years. The first tumor is apt to be in the lower colon and the others to occupy sites progressively higher, which excludes the possibility of implantation. The time interval between the appearance of the tumors is too long to encourage their interpretation as delayed recurrence even were their sites of development to favor this explanation. The tumors start on the mucosal surface and frequently do not penetrate the bowel wall deeply, which excludes metastasis by the blood or lymph stream or peritoneal fluid. Finally, in such a colon polyps are very apt to be found, and their known tendency to carcinomatous change seems to offer adequate explanation of the circumstances of such a case.

Appreciation of the surgical problems of polyposis of the colon is, then, of very considerable importance to the surgeon. This matter has been discussed more or less in the literature, but recognition of the cancer tendency in polyposis of the colon is of relatively recent date. The subject has been discussed by Babler, Niebruegge and Fisch,[12] Lilienthal,[13] Lockhart-Mummery,[14] Bardenheuer,[15] Back[16] and Forster.[17] It is a benign condition with definite carcinomatous tendencies—in other words, a clear precancerous lesion and this fact will perhaps largely determine our final position with reference to treatment. At present there is no generally accepted plan, and the suggestions have ranged from simple excision of the polyps to complete colectomy, which, radical though it seem today, may yet come into quite general usage. This subject would well repay careful study.

In a practical sense, then, we must recognize that cancer of the colon may occur as multiple primary lesions, that an entirely similar picture may be produced by tumors which develop secondarily, and that polyposis of the colon has a very definite tendency to malignant degeneration.

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CLOSURE OF AN ARTIFICAL ANUS OF EIGHT YEARS' DURATION, WITH SOME REMARKS ON THE QUESTION OF INTESTINAL ANASTOMOSIS

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Instances of the closure of an artificial anus of more than one year's duration are obviously rare. During that interval no material depreciation in the function of the intestine below the abnormal orifice has taken place. The closure of the opening leads immediately to the complete restoration of colonic action. On the other hand, in cases of long standing in which the entire bowel contents are discharged through the artificial anus it is quite reasonable to infer that, owing to the disuse extending over a period of years, the motor, vascular, secretory and nervous apparatus of the intestinal wall may have become so atrophied or otherwise changed that even a partial restoration of their several functions may not take place when the abnormal orifice is closed.

In 1913, the writer was asked to see a patient who had developed an artificial anus following an operation for the relief of an infected appendix. As a fecal fistula, especially where the appendix at the junction with the cecum happens to be gangrenous, is not a rare postoperative complication, and as these fistulæ usually close spontaneously in the course of ten days to several weeks, conservative measures were advised, notwithstanding that the entire bowel contents passed through the artificial opening. As the condition remained unchanged at the end of a year, a second consultation was held to discuss the propriety of operation. The patient, about sixty years of age,

had for many years been an incurable paranoiac and required the constant attention of two nurses, whose testimony, together with that of the attending physician, conclusively showed that the occasional fecal discharge through the abnormal opening did not cause the slightest perceptible annoyance or discomfort, and that the usual prophylaxis prevented any irritation of the adjacent skin. Furthermore, the relatively low position of the opening precluded any deterioration in the patient's general condition. While closure would unquestionably have been attempted in an otherwise normal subject, the fact that such a procedure, if successful, would, in the case of an incurable paranoiac, have neither added to her comfort nor have removed the need of constant nursing, seemed to justify a *laissez-faire* policy. This conclusion was still further strengthened by the surmise that a marked decrease in reparative power, due to the long-continued mental derangement, would, if present, unfavorably affect the chance of successful closure, if it did not actually predispose to a fatal postoperative peritonitis.

After several years, prolapse appeared. This at first was slight and easily controlled by an overlying pad, although it gradually increased in size. After a time, however, the prolapse, always reducible, became more complete, until finally, seven years after the original operation, it formed a voluminous mass, balloon-shaped, bulging over the side of the patient for a distance of at least twelve inches, consisting evidently of the entire ascending colon. When reduced every contrivance failed to prevent its spontaneous return. In this condition it became both a source of annoyance and irritation, and its resection seemed justifiable, provided that the capacity of the distal gut to function could be established with reasonable certainty. Would the delicate nervous, secretory, vascular and muscular mechanisms of the wall of the large intestine, after so many years of inactivity, respond to the stimulus of intestinal contents and conduct them by their successful coördination through the entire length of the large intestine to and through the rectal outlet?

Digital examination disclosed a tonic rectal sphincter. Ene-

mata were expelled, the first with a considerable discharge of mucus and feces, the first in seven years. A small quantity of an aniline dye, administered under low pressure in a colon irrigation, appeared at the artificial anus. The operation, as suggested, was therefore performed, the terminal ileum, the entire ascending and several inches of the transverse colon being resected through an incision enclosing the former operative scar and the artificial anus, followed by a lateral anastomosis. The wound was closed in layers around a protruding drain. The patient stood the operation well. The temperature, never above 100°, was normal on the fourth day. There was no distention at any time. Peristalsis was quickly reëstablished, gas being passed per rectum at the end of the first twenty-four hours. The bowels moved naturally on the second day without enema or drip. For the first week the bowel movements were fluid and occurred on an average of once every four hours. The buttocks became considerably excoriated, necessitating constant watching. A small, occasional dose of morphine lessened the frequency. The first formed movement occurred one week after operation, and afterward the consistency varied. The patient took fluids and soft nourishment with relish. The superficial wound became infected, with some sloughing of the aponeurosis. A persistent sinus remained.

During the past four years the patient's condition has been satisfactory, although the postoperative sloughing of the aponeurosis was followed by some bulging in the scar. The function of the bowels is normal.

The type of anastomosis after intestinal excision presents a most interesting question and a fruitful theme for discussion. In the small intestine, end-to-end anastomosis by suture, the abdomen being closed without drainage, gives excellent results. In the large intestine a similar procedure may be followed in the sigmoid, of which the mesentery ensures proper peritoneal adaptation and protection. Furthermore, in this location a flexible rubber tube inserted into the rectum by an assistant and directed by the operator's hand through the site of anastomosis into the bowel above it serves to conduct gas and the

colon contents through the sutured segment, thereby averting the danger of possible local distention and leakage. In other parts of the large intestine, where proper peritoneal covering cannot be secured, the writer prefers a side-to-side anastomosis after closure of the divided intestinal lumina. This seems to afford the greatest protection against subsequent leakage with the formation of a fistula, of which the successful closure is frequently extremely difficult. The writer has had no or little experience with end-to-side anastomosis. Theoretically, after excision of the ileocecal junction, this method of anastomosis is supposed to restore more satisfactorily than a side-to-side anastomosis the normal condition of this part of the intestine. In informal discussion, however, with those experienced in this method of anastomosis, the writer has gained the impression that both leakage and postoperative obstruction from undue angulation or adhesions are more common than after other methods of anastomosis. Perhaps the standardization of the most desirable method of anastomosis is impossible. The personal equation may prove, after all, the determining factor, the choice of method depending upon the individual skill and preference of each surgeon.

The writer advocates drainage with a small, flexible, rubber tube, enclosing a wick of gauze, in all cases of anastomosis involving the large intestine. Frequently the wound remains free from infection and the drain is permanently withdrawn at the end of twenty-four to seventy-two hours. Occasionally infection appears in the abdominal incision while the intestinal repair is prompt and satisfactory. In a few instances a small, fecal fistula forms, a possibility that fully justifies the use of precautionary drainage. It is scarcely necessary to add that all contact of the drain with the site of anastomosis should be carefully avoided.

While it is both impossible and undesirable to urge any special method of treatment from the experience of a single case, the result, in the present instance at least, has proved sufficiently encouraging to justify an attempt to close an artificial anus, even though it be of long standing and complicated by conditions of impaired reparative power.

DISCUSSION ON THE PAPERS OF DRS. DEAVER, PECK, JUDD, MILLER AND ELIOT

Dr. Carl A. Hamann, Cleveland, Ohio:

In connection with these papers I wish to state that I have had 49 resections of the large intestine, including the rectum, in the past six years, with a mortality of 3, or a little over 6 per cent. The mode of death in one of these cases was interesting to me. In order to remove all the carcinomatous lymph nodes in a case of carcinoma of the cecum, we had to cut pretty far over toward the median line and we injured a large vein, possibly the superior mesenteric. It was tied in two places and the patient progressed for the next few days without any particular symptoms. She, however, died on the twelfth day from repeated intestinal hemorrhages of large amount. I imagine that in this case death was due to a mesenteric thrombosis, a very unusual mode of death in my experience.

The other two deaths were the result of perforation of a distention ulcer. These distention ulcers, as described by Kocher some years ago, occur above the site of the obstruction, may rupture, and this results in peritonitis.

I should like to hear Dr. Deaver's views on the subject of mobile cecum, whether or not this is responsible for right-sided pain and whether or not cecopexy is a desirable operation.

Dr. W. W. Keen, Philadelphia, Pa.:

I would like to say a word about two of the papers. The first one is that of Dr. Judd on "The Surgical Treatment of Diverticulitis of the Large Intestine." There is nothing like having been a patient yourself, provided you have escaped the undertaker; and as to that all that is necessary is to say that my good friend, Dr. William J. Mayo, was my surgeon.

In the spring and summer of 1911 I had a persistent diarrhea, mostly nocturnal, without any pain, no tenesmus, or anything of that sort. I went to Europe in the early summer, and in September, von Eiselsberg, an old friend in Vienna, examined me and found in the left lower abdomen a tumor. I was approaching seventy-five, and I said to him, "I suppose without much doubt that is a carcinoma of the large bowel?" He shook his head and replied, "Well, it gives cause for reflection." He was no more definite than that. Within fifteen minutes after he left I was writing to Dr. Mayo, saying I was coming home soon and wished to place myself under his care as quickly as I

could. After he examined me he said, in substance, "I hardly think that is a carcinoma, but I am not willing to make a positive diagnosis." He operated upon me and found two diverticulæ of the sigmoid, each of which contained a small coprolith. One of them had ulcerated through, but, fortunately, before it had penetrated entirely had created adhesions about it and the coprolith had escaped from the diverticulum into this little mass of adhesions. There was only a drop or two of pus. In a little while that would have penetrated and escaped into the abdomen, and very likely a fatal peritonitis would have followed. The other one was so thin in its wall that I was rather astonished that a false step, or anything that would produce a sudden jar, had not ruptured it.

Dr. Mayo's operation consisted in removing a V-shaped portion of the bowel and then joining the two ends together. You see the result before you after thirteen years next November.

I suffered gas pains for a few days, but on the third day, I remember very well, I was sitting up in bed with a pad on my knee and writing a letter, and from that time to this I have never had the first twinge of pain. Preceding the operation I never had the slightest pain even in connection with the ulceration of the bowel by the little coprolith. While I was in Rochester, my son-in-law, the late Dr. Walter J. Freeman, was asked by one of his friends what was the matter. Dr. Freeman had a great deal of dry humor about him, and he described the pathology of it in the argot of the automobile, "As I make it out it was a blow-out of the inner tube!"

The other subject is the artificial anus. I operated on one of the very worst cases of carcinoma of the rectum that I ever saw, requiring removal of the sphincter muscle and about twelve inches of the rectum. The growth was very nodular and hard. I had previously made an artificial anus in the left iliac fossa. I did not close the perineal opening, but left that open also. After two or three years it annoyed the patient so much by prolapse that he wanted it closed, which I did. That left, of course, only the artificial anus in the inguinal region. All of the contents of the bowel escaped from it. He lived for over twenty years and then died from a sudden self-reduction, *en masse*, of a strangulated hernia. I knew nothing of it for nearly a week. I asked one of my assistants to see him (he lived out of town), but he was then moribund.

What I wish to emphasize is the *mode of dressing of the artificial anus*. All of you know, by an uncomfortable experience with a great many patients, how annoying any artificial anus is. There is no apparatus that I know of that can retain the feces by application that does not become foul and ill-smelling, compelling the patient to lead almost a

hermit existence because he is the center of so much that is disagreeable. This patient I spoke of was a graduate of Yale and a very intelligent man. For twenty years he conducted a large wholesale business and went to- and- fro wherever he wished. He never had any return of the disease in that long period, which was most gratifying.

He devised a method of treatment of his artificial anus which was absolutely perfect. I have repeatedly sat close to him in my office and not once did I detect even a whiff of anything that was disagreeable. The only time that he had to remain at home for a day or two was when he had a diarrhea with an exceedingly liquid stool. Then the apparatus did not work.

You will find in the very last chapter of the last volume of Keen's *Surgery* (Volume VIII) an account of his method of dressing an artificial anus. Less than thirty days before his death I had him go down to a photographer, take all dressings away and then apply his dressing, which was simplicity itself. All that is needed is some ordinary absorbent cotton, a little bandage and a truss to hold the pad in position, even if your patient does not have a hernia. A very simple affair after all. You will find six or seven photographs showing the different steps of the dressing. I am sure it will be a boon to any of your patients and a comfort to you if you will look at that chapter and have any patient adopt that method of dressing it.

Dr. George E. Brewer, New York:

I wish to say a word in regard to the difficulty that surgeons have in making a diagnosis of carcinoma of the colon. This has been brought forcibly to my attention by two patients, who were both about the same age, both with practically the same symptoms and both having had a history of a recent gain in weight. The first patient had suffered from intestinal disturbance for many years, evidently a chronic colitis; there had been a good deal of indigestion; she was troubled considerably with gas, and at one time had an attack of pain which suggested an acute cholecystitis or acute gall-stone colic. A week later her gall-bladder region was distinctly tender, and I made a diagnosis of cholecystitis; but before operating, suggested that she have a series of *x*-ray plates made of her colon. The *x*-ray plates revealed a definite stricture of the transverse colon something like this (blackboard demonstration). As soon as we saw this we immediately were suspicious of carcinoma. The most careful palpation failed to reveal anything. The series was repeated and still the picture was the same. On further examination by the fluoroscope an attempt was made to force the barium solution back from the distal portion through the strictured area.

There was no change. Other plates were made, so that she had thirty or forty plates, and the pictures were the same, except in one instance, and that was the small, narrow, connecting link dilated to perhaps a quarter of an inch, but only for a short distance. I felt quite sure that this patient had a carcinoma of the colon and suggested an exploratory operation. On operation the colon was found contracted, but its walls perfectly normal. By closing the distal portion and compressing the proximal portion we were able to dilate the contracted area to the size of the sigmoid. It was evidently a spasm. She has never had any symptoms since the operation.

The second patient I saw six or eight weeks after the first one reported. A woman of the same age, with a history of a gall-bladder operation eleven years ago when several stones were removed. Some four or five years later she had a single attack of gall-stone colic, was well for four or five years and then had another attack, and then the gall-stone colic apparently disappeared. For five or six months she had occasional symptoms of peristaltic unrest, occasionally a distinct pain, which might be called a colic, which would last for a few hours, and which was relieved by cathartics. The *x*-ray pictures were the same. It was difficult to recognize such pictures as belonging to the one patient or to the other. I did an exploratory operation, found a carcinoma of the transverse colon, did an extensive resection, and she did well.

With reference to the question of diverticulitis, I was interested in Dr. Judd's paper, and I was struck with the fact that the great majority of our cases are not chronic but acute.

When this question was first brought up fifteen or twenty years ago, Dr. Mayo read his paper on the chronic form, which was at the same meeting that I presented 5 cases illustrating the acute form. The two types are distinct. The acute cases, as Dr. Judd has said, are often spoken of as the acute appendicitis on the left side of the abdomen, and I wish to emphasize that this distinct type is a surgical emergency, exactly in the same way as is the acute appendicular infection.

My experience is limited, but I can refer to 7 cases that have come under my observation which belonged to the acute type. In 1 case the duration was only three or four days. It began like an acute appendicitis—abdominal pain, nausea, vomiting, subsequent rise of temperature and a gradual increase in rigidity until we had a picture of an acute peritonitis. At operation I found a perforated diverticulum of the sigmoid with the concretion lying in the floor of the pelvis with a general peritonitis. This patient died.

Another case with exactly the same symptoms, but in the midline;

I made a median incision and found a perforation of a small diverticulum, giving all the evidences of an acute appendicitis.

The other acute cases were all distinctly on the left side.

Dr. Charles H. Mayo, Rochester, Minn.:

I was very much interested in the paper on multiple primary malignant foci of carcinoma. If there is any place in the body where we can have malignant foci of that type it is in the colon. Whatever happens to the colon the whole area is subjected to the same influence. Multiple foci probably have a lot to do with the infection and the causation of diverticulitis.

In operating for diverticulitis a most remarkable peristaltic action may be noted. These waves pass along the transverse colon about four inches apart; the first stops in severe spasm, the next keeps on and almost bursts the intestine.

Diverticulitis seems to be increasing, particularly among highly civilized people, and with the more scientific preparation of foods. Diverticula do not exist among the people who live the ordinary primitive type of life. They rarely occur under fifty years of age.

Occasionally a spasm occurs of the kind reported by Dr. Brewer. It may be a spasm instead of a diverticulum. It may last for hours. What is to be done about it? Are we going to change our diet or are we going to carefully watch the chemistry of the large bowel?

Dr. Deaver's point is a good one. I sleep a good deal better if in operating I make a safety-valve gas vent of the closed end of the bowel, about the size of the thumb nail. If there is to be trouble from gas pressure it will not come the first day; it will come the night of the second day and the third day.

If you use the Murphy button in these cases and suture end-to-side, every fifth or sixth patient will have dilatation, which produces tension, and it is tension on the suture line that is fatal.

Dr. Willy Meyer, New York:

Dr. Judd's mention of a case of perforated diverticulum into the bladder recalls to my mind an observation I made a few years ago. After a barium clysma given out of town a spontaneous abscess had developed close to the left anterior superior spine. A few days after incision a fecal fistula formed. Establishment of an artificial anus on the right side was now indicated. Before this could be done a second diverticulum perforated into the bladder. The patient now had two openings from the intestinal portion carrying the diverticula. Later the proposed artificial anus was formed by his surgeon at home. He developed thrombosis of one of the iliac veins and died from embolism.

We certainly should not place too much reliance on the *x*-ray report. I had one patient with distinct clinical symptoms of colonic obstruction. A barium clysma was given and the report returned that there was no stricture. Nothing pathological had been found, and yet the patient had shown typical stiffening of the intestine, etc. Against my advice she returned under the care of the medical man. Two weeks later she developed all the signs of acute intestinal obstruction, which necessitated immediate operation in two stages. At the second stage a carcinoma was found in the middle of the transverse colon. The radiographist explained that the clysma had distended a colonic haustrum proximally and distally to the existing stricture. Both joined hands and overshadowed the small tumor. In such conditions I believe that our clinical signs should be the first to guide us.

I had a patient with resection of the lower end of the ileum, cecum and ascending colon with a portion of the transverse colon for carcinoma. She made a good recovery. The pathologist reported adenocarcinoma. Several years later she developed a bleeding cancer of the bladder. This was also excised and she lived for more than twenty years, when she died from acute pneumonia. Such observation shows how comparatively benign these cancers are.

A word yet with reference to the formation of an artificial anus or a cecal fistula in cases of acute complete or incomplete intestinal obstruction. Within the last six years I have done the following operation five times. I think in cases of colonic obstruction it is wise to make a cecal opening. Under local anesthesia and with the help of a McBurney intermuscular incision the cecum is pulled forward, stitched in place and then, according to necessity, twenty-four or thirty-six hours later, punctured with the pointed Paquelin cautery. A rubber tube with a number of holes at the end is then inserted and easily retained in place for eight, ten, fourteen or more days, while one will have an absolutely watertight fistula, which closes spontaneously later on.

Dr. Joseph A. Blake, New York:

This is an extremely interesting topic, and I wish to mention a sign in localizing the site of an obstruction in the large intestine. I do not know how much attention has been called to it. I was reminded of it when one of the speakers mentioned rigidity of the gut and its disappearance. The fact that the cecum looms up and then disappears under the hand does not always mean that the growth is in the neighborhood; it may be in the sigmoid flexure. The best sign that I have found to determine the site of the obstruction is the site of the loudest bubbling of gas. As the advancing peristaltic wave proceeds toward the obstruc-

tion, the gas is not crowded through the obstruction but is compressed until the constriction of the peristaltic wave gives way and the gas rushes backward through it in an oral direction. The obstruction lies not far aboral from the point at which the bubbling is heard. This sign often enables one to locate the growth, and in a number of cases, if the growth is located, no matter if there is quite marked distention, the incision can be made over the growth and the mass removed by the two-stage von Mikulicz operation without the necessity of a preliminary artificial anus. Such operations usually are very successful even if the obstruction has been complete and of long-standing.

In regard to diverticula and diverticulitis: As a result of my own experience, I have tentatively separated these cases into three groups: The first, those cases in which the diverticulum has ruptured suddenly with the production of more or less diffuse peritonitis. In a second group there has been the slow production of a localized peritonitis, often with the formation of a large mass. The third type, of which I have seen more in the last two or three years, is that in which there are a number of diverticula grouped along a considerable length of the intestine, often from the splenic flexure down to the sigmoid flexure, that is, involving almost the entire descending colon. As a result of inflammation of the closely lying diverticula, extensive fibrosis of the walls of the intestine may occur and even obstruction.

These cases are interesting on account of the difficulty of operation. I have found in such cases the lumen of the gut so small as to make anastomosis almost impossible. If we attempt to remove all the constricted portion of intestine it may necessitate removing the entire descending colon, which means mobilizing the splenic flexure and adding materially to the danger of the operation. In one patient, in whom I resected the lower portion of the descending colon for obstruction caused by diverticulitis, the lumen of the remaining portion of the descending colon was so small that I could only introduce my little finger, yet I was able to do a von Mikulicz operation successfully.

With regard to the etiology: Instead of being due to pressure produced by violent peristalsis, I believe diverticula are due to atony of the gut, and that they are really herniæ of the mucosa between the overstretched and separated fibers of the muscularis. We rarely find them in cases of obstruction.

Dr. J. J. Buchanan, Pittsburgh, Pa.:

Dr. Deaver and Dr. Peck have both called attention to the very great difference in prognosis after excision of the ileocecal coil and of the sigmoid flexure, and that has been attributed by one of the gentlemen

largely to the increased bacterial contents of the bowel below. Personally, I think it is due to a mechanical condition; the mechanical arrangements of the bowel on the right side of the abdomen are much better for anastomosis than they are on the left side. On the right side you can excise the entire ascending colon, mobilize the hepatic flexure, if necessary, and then you have any quantity of small intestine and any quantity of transverse colon at your disposal for anastomosis. The question has been brought up as to what is the best way to anastomose the small intestine to the transverse colon. It has seemed to me that in the cases on which I have operated this depends on the conditions present. If you have much distention, as you often have of the small intestine, you can make an end-to-end anastomosis with the transverse colon without much frilling, and that is the ideal method; but if the lumen of the small intestine is normal, you should do as Dr. Charles H. Mayo does—make an end-to-side anastomosis, which seems to be almost equally as good.

Dr. Peck has called attention to a case which is typical of many cases which we all see; a case in which, without previous symptoms, he finds a carcinomatous stricture in the large bowel, so that he has only a lumen the size of a lead-pencil. This condition comes on so slowly, over a period of months and often years, that the patient does not notice the increasing constriction.

I wish also to comment on the procedure which has been referred to occasionally here, and that is the making of a temporary fecal fistula above the site of an end-to-end anastomosis. This is a safety-valve which is used, I believe, by Dr. Ochsner and in the Mayo Clinic. Personally, I always feel better if I have a small rubber tube anchored above the site of my anastomosis, so that gas cannot break through the stitches, as Dr. Mayo has described.

With reference to an artificial anus on the right side, I would say that my experience has been that the patient is in a very much more unfortunate condition than if he has the artificial anus on the left side so far as the contents of the bowels are concerned. Dr. Keen has very graphically described the measures of caring for an artificial anus. It seems to me, however, the question of whether a patient is comfortable after an artificial anus on the left side is a question of that patient's diet and habits. So far as my own patients are concerned, I can only quote the French woman who said she wondered why the "Good God had not made everyone's anus in front."

I would like to call your attention to this instrument (exhibits it), which is a modification of Dupuytren's clamp that I have used with great satisfaction. The external part lies on the abdominal wall and

does not protrude like forceps; it has a screw which can be absolutely regulated to one-tenth of an inch, and if it is uncomfortable it can be loosened a little. I do not know of any other instrument that has been devised or published which will allow of less protrusion from the abdominal wall, and the pressure of which can be absolutely governed to the finest degree.

Dr. Emmet Rixford, San Francisco:

I would like to report two cases of diverticulitis of the sigmoid, in both of which the patient died of peritonitis, one from spontaneous rupture (nothing more untoward had happened than stubbing his toe); the other from rupture brought on by the administration of a dose of castor oil given as a preliminary to a proposed operation.

In the matter of etiology some support is given to the doctrine of Hansemann, that the herniation commonly occurs through intervals in the muscular coat which normally give passage to the bloodvessels, by the fact that in a number of cases we have found diverticula occurring in appendices epiploicæ.

Dr. Leonard Freeman, Denver, Colo.:

In connection with Dr. Miller's paper, I desire to report an instance in which three separate primary cancers of the colon appeared in the same individual at different times.

The case was that of a white man, of American ancestry, aged fifty-two years, who consulted me for an aggravated intestinal obstruction October 27, 1921.

A preliminary cecostomy was done immediately, followed, in a short time, by a laparotomy, which revealed a hard, ulcerated, circular carcinoma the size of a golf-ball, constricting the transverse colon not far from the splenic flexure, and with many enlarged mesenteric glands. The growth was widely removed, together with the larger portion of the transverse colon and its mesentery, a side-to-side anastomosis being done and the abdomen closed without drainage. He made a good recovery. The remainder of the colon appeared normal. There were no evidences of polypi.

Six months later he returned with intestinal obstruction again. I reopened the old cecostomy and somewhat later did an abdominal section, expecting to find a recurrence of the original tumor or at least a stricture of the anastomosis. Instead of that, I found the anastomosis in good condition, but a new, hard, annular, ulcerated cancer of the colon, about six inches proximal to the anastomosis, and almost exactly like the old one. It evidently had begun on the inside of the bowel, the peritoneum not being involved.

The growth was widely resected, an end-to-end anastomosis done and the abdomen closed without drainage. The old anastomosis was not disturbed. The recovery was uninterrupted.

Twenty-one months later the patient again returned, because of failing health and strength. There was no intestinal obstruction, but much distress in the rectum. An examination revealed a large, indurated, ulcerated, immovable cancer of the rectum, which could easily be felt with the finger, and which was manifestly inoperable. There were no signs of trouble in the colon.

The pathological examinations of the first two tumors were made by Dr. Helen Craig and showed them to be papillary carcinomata. No pathological examination was made of the rectal tumor, for obvious reasons, but its nature was clinically beyond question.

For a long time, multiple cancers of the colon have been reported at intervals, but the explanation remains obscure. The possible explanations are:

1. That they are entirely separate growths.
2. That they are metastases through the lymph or blood.
3. That infective material is carried through the peritoneal cavity.
4. That particles of cancer tissue floating free in the lumen of the bowel are grafted into some abrasion of the mucosa.

I believe the last-named theory to be the correct one, at least in the case just reported. The colon is peculiarly adapted to such autografting, owing to the stagnation of its contents and to the liability to traumatism from hard and rough material in the feces. It is worth considering that the second cancer, in my own case, may have found a localizing spot where the rubber-covered forceps were applied to the bowel proximal to the first growth—at any rate, it occurred at about that point.

For anatomical reasons it is not likely that metastasis could take place through the blood or lymph; while the peritoneum is excluded as a carrier by the facts that in the one instance it was not involved and in the other the growth was in the rectum, where there was no peritoneum.

Dr. Alexis V. Moschcowitz, New York:

The great frequency, if not exclusiveness, of diverticulitis on the left side has so frequently been emphasized that I may be permitted to mention that I have had occasion to operate at least a half dozen cases of acute appendicitis which were caused by a perforated diverticulum of the appendix. In a further case I had no hesitancy in making the diagnosis of an acute suppurative appendicitis both from physical

signs and symptoms. The operation, however, revealed a perfectly normal appendix (which was removed), and a perforated diverticulum of the cecum was found to be the cause of the peritonitis. Several years later the patient again returned with similar symptoms. *X*-ray examination revealed numerous diverticula in the cecum. All symptoms disappeared under expectant treatment.

Dr. Daniel F. Jones, Boston, Mass.:

I am presenting a small group of end-results, because I understood that the symposium was to be on end-results.

TABLE I.—CARCINOMA OF THE COLON

	Number of cases.	Deaths.	Mortality, per cent.
Resections	33	6	18
Short-circuiting	16	1	6
Exploratory laparotomy	5	2	40
Colostomy	12	0	0
Totals	66	9	13

TABLE II.—CARCINOMA OF THE COLON

	Number of cases.	Deaths.	Mortality, per cent.
Left colon	27	5	18
Right resections	6	1	16
Total	33	6	18

TABLE III.—END-RESULTS, RESECTION OF THE COLON

Total number of operations	33
Total number of deaths	6

	Number of cases.	Total number of cases, per cent.
Living	21	63
Living three years or more	14	42
Living less than three years	10	
Living five years or more	5	21
Living less than five years	16	
Total	66	

TABLE IV.—END-RESULTS, RESECTION OF THE COLON

	Number of cases.	Tota number of cases, per cent.
Operated upon three years or more	14	
Operative mortality	6	
Died within three years	3	
Total living and dead at end of three years	23	
Percentage living three years		61

TABLE V.—END-RESULTS, RESECTION OF THE COLON

	Number of cases.	Total number of cases, per cent.
Operated upon five years or more	7	
Operative mortality	6	
Died within five years	4	
Total living and dead at end of five years	17	
Percentage living five years		41

There are three fundamental principles in resections of the large intestine which should always be kept in mind:

1. Relief of obstruction before attempting resection.
2. Perfect walling off of the field of operation.
3. Some method for the prevention of intracolonic pressure after resection.

To accomplish the first and third objects a cecostomy by the Gibson method—that is, the inversion of the cecal wall about a large tube, a half inch in diameter—has been the most satisfactory in my hands when the cecum has not been removed. It gives a clear field to operate in. It relieves obstruction and it prevents intracolonic pressure after operation.

Dr. Rudolph Matas, New Orleans, La.:

Since the discussion on the surgery of the intestine has become general, I will take up a brief moment to illustrate on the blackboard the method that I have found most serviceable in dealing with circular or end-to-end anastomosis of the small bowel. In dealing with the neoplasms of the colon I have resorted to the Mikulicz three-stage operation more often than any other. But the technic must vary with the part of the colon involved; especially is this true of the extirpation of the splenic angle. In a general way I am more and more convinced of the value of a preliminary artificial anus in adding to the safety of any radical extirpation of the colon. As the time is too limited to allow for a discussion of the various phases of colonic surgery, I only wish to call attention to the added security and comfort given to a circular suture of the small bowel by the addition of lateral anastomosis made 8 or 10 cm. higher up with a small Murphy button. As you see by this diagram the end-stumps are thoroughly united by a double row of mucoserous sutures put in the usual way (Connell principle preferred) and again laterally by the Murphy button. An important feature of the technic is that each half of the Murphy button is slipped into the lumen of the gut through each one of the open ends before the terminal

suture is effected. When the ends of the gut have been securely united, regardless of the narrowing of the lumen at the suture line, the buttoned halves are readily clamped together after exteriorizing the projecting stems through a small button-hole incision cut over the stretched intestinal wall. After the button is tightly clasped the button area is still further protected by a circle of continued subserous sutures.

The advantages of this procedure are: (1) That it allows the terminal circular suture to be made absolutely secure, even though the bowel lumen may be contracted, by the thorough apposition of the bowel ends; (2) that the fecal and gaseous circulation of the bowel is ensured by the lateral communication made above it, thus relieving the terminal suture line of all strain from overdistention by gases or feces. One of the immediate effects of a circular enterorrhaphy is to cause an inflammatory edema of the line of suture, which is stenotic or obstructive in its effect. This again is followed by a distention of the paretic bowel on each side of the line of suture, which is more likely to occur with the greater thoroughness with which the divided ends have been sutured. When a supplementary lateral anastomosis is made with a button by the simple method shown here, all the intraintestinal pressure is relieved, the fecal circulation is not interfered with and the terminal suture line is allowed to heal without overstrain from gaseous distention.

Since adopting this additional supplementary lateral anastomosis with the button, I have felt that the safety of the patient had been increased and the convalescence was more speedy and comfortable.

Dr. Deaver (in closing):

I have nothing to add to this paper, and I agree *in toto* with all that has been said.

In answer to Dr. Hamann's question, I have to say that I have never had the courage to operate on a mobile cecum.

Dr. Peck (in closing):

In connection with these papers I recall a case operated on a year and ten months ago—a man aged seventy-five years, with a large carcinoma of the splenic flexure. It was removed by the method described by Dr. Miller, and he made an excellent recovery. Nineteen years previously he had a carcinoma of the sigmoid flexure removed by Dr. Dowd, from which he made an excellent recovery. At the time of the latter operation no sign of the site of resection could be distinguished on the sigmoid or on the lower descending colon. The new growth was at least eight or ten inches above the site of the former

resection. I do not believe it was a secondary metastasis, but quite an independent second carcinoma with an interval of nineteen years between the two growths.

With reference to the fact that glandular swelling may not be malignant, short-circuiting was done in a case of carcinoma of the transverse colon in a woman, aged seventy years. It was adherent to the posterior parietes. There was a fistula which drained for a long time, and finally an evaginated loop of gut protruded through the opening. At the apex of the extruded loop was a round carcinomatous ulcer. The carcinoma gradually protruded through the opening, and finally, after it had come out five or six inches, it was cut off, the cut ends were sutured and recovery followed. She lived for two and a half years with a wide-open fistula. Later, as the result of an extensive resection, she died from postoperative peritonitis.

Dr. Judd (in closing):

My purpose in reviewing this series of cases of diverticulitis was to call attention to the frequency with which the condition is recognized as compared to only a few years ago and the results accomplished by modern methods of treatment. Undoubtedly, the efforts of the men of this Association are largely responsible for the progress that has been made in the handling of the disease. So far as I have been able to determine the first report of a series of chronic cases was made by Mayo, Giffin and Wilson in a paper before the American Medical Association in 1917. At the same time Brewer presented a series of cases of acute diverticulitis with abscess formation. Since then many cases have been reported in the literature.

Another point that I wished to emphasize in presenting these cases was that many of them can be treated very satisfactorily by conservative measures, especially if they come for consultation reasonably early. Resection is attended by a high mortality even with the best efforts, and the possibility of accomplishing the desired result by other means should be excluded before it is attempted.

Dr. Eliot, Jr. (in closing):

The paper to which Dr. Miller referred, "Recurrence *versus* Metastasis in Carcinoma," was read before this Association two years ago. As Dr. Matas has suggested, there is no viscus in which a fresh outbreak of carcinoma is more likely to occur than in the large intestine. In the paper quoted, I referred to a patient in whom two and a half years after the removal of a carcinoma of the sigmoid a second carcinoma appeared at the splenic flexure. This patient is still living almost

three years after the second operation, without sign of further recurrence. I would also like to refer to a second patient in whom the splenic flexure was removed, about five years ago, followed by a side-to-side anastomosis. Four years after the operation a second carcinoma appeared in the lower part of the sigmoid or upper rectum, so low that it was difficult to make a palliative anastomosis. There was extensive liver metastasis. It is of interest to note that the patient's condition after the anastomosis was less comfortable, owing to the passage of repeated and irritating intestinal contents through the rectum than would have been the case after an artificial anus. In both cases it is reasonable to infer that the second carcinoma was in no way connected with the primary carcinomatous focus.

In connection with Dr. Judd's paper, I would like to call attention to the possibility of foreign bodies causing an acute diverticulitis in the sigmoid flexure. I refer to one such instance in which the patient presented the symptoms of a virulent peritonitis of sixteen hours' duration, due to a perforation, the size of a slate-pencil, in the center of an indurated mass in the wall of the sigmoid. Efforts to close the perforation with a purse-string suture were unsuccessful, owing to the friability of the tissues. Neither was the omentum of sufficient length to be brought down and sutured so as to close the abnormal opening. It was, furthermore, feared that the closure of the perforation by the approximation and suture of the small intestine might lead to some form of postoperative obstruction. The sigmoid was therefore sutured with some difficulty, owing to the short mesentery, to the parietal peritoneum, leaving the perforation extraperitoneal and so establishing a fecal fistula which could later be closed if it refused to close spontaneously. The fistulous tract, however, persisted with a small discharge, although four months after the operation a fish-bone at least an inch in length, which was not apparent at the time of the operation, was discharged. Still persisting, as is not unusual with fistulæ communicating with the large intestine, the patient was ultimately relieved by a resection of the affected part of the gut.

INTRA-ABDOMINAL RUPTURE OF INTESTINE FOLLOWING STRANGULATED FEMORAL HERNIA

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That better understanding of the abuse of taxis and the crime of delay in strangulated hernia, together with the early recognition and prompt surgical intervention, have diminished the frequency of bowel gangrene and its direful sequelæ.

Many valuable contributions are found in the earlier literature telling the story of delay and describing intestinal "wet death" within the hernial sac, but in general there is little information to be found bearing upon that more serious complication mentioned in the title of this paper.

A. J. McCosh,[1] in 1889, collected and tabulated 150 cases, for which immediate resection was done. C. L. Gibson's[2] statistical study, based on "A Thousand Operations for Acute Intestinal Obstruction and Gangrenous Hernia," supplied a need at that time which had hitherto not been met. Neither of these interesting communications makes mention of an intra-abdominal rupture of the constricted gut.

A reference of historical interest from the writings of the English anatomist, John Hunter,[3] bearing on this subject, which reflects his own wide experience, is worth quoting here. He writes:

"It is very curious to observe in hernias, that while the gut is in the sac and alive no inflammation takes place within the sac or integuments; but the moment the gut becomes mortified or dead, the stimulus of an extraneous body takes place immediately; an outlet is then endeavoring to be made by the inflam-

mation and suppuration of the sac, forming an abscess in it; which matter, with the contents of the gut, is brought to the skin. While this is going on, the sound gut within the abdomen, where it passes into the rings, adheres to those rings all around; so that when the abscess is formed, burst or opened, and the mortified parts sloughed off, these ends of the gut open into the abscess, and not into the cavity of the belly."

In an effort to ascertain the frequency of a complete severance of the gut at the point of constriction within the abdomen an abstract of the author's findings was sent to Sir Berkeley Moynihan, who courteously replied:

"I have had a number of cases of strangulated hernia in which the intestine has given way at the line of constriction. It has happened more often with femoral than with inguinal hernia. In their treatment, as a rule, the intestine is pulled down into the hernial sac, a resection made and an end-to-end anastomosis by suture completed. We have notes of 3 cases in which a fecal fistula was formed by merely laying open the adherent intestine in patients extremely ill. In 2 of these closure occurred after the spur between the two limbs had been destroyed by pressure. We have no note of any case in which fecal extravasation occurred into the peritoneum, as in your case."

W. B. Coley, whose wide experience at the Hospital for Ruptured and Crippled is well known, in a personal communication writes:

"From my own experience, as well as my knowledge of the literature of hernia, I know of no case quite similar."

It is reasonable to assume that accidents of this character have been observed before; however, the literature available contains the record of but 1 case clearly belonging to this category. This was reported by T. H. Manley[4] before the Pan-American Medical Congress, November, 1896.

Case I.—(Manley's case.) Male, Chinaman, aged twenty-eight years. No previous history of importance except a reduc-

ible inguinal hernia of the right side of eight years' duration. No truss had been worn for three months. One morning the hernia descended and was more painful than usual; repeated efforts failed to reduce the mass. Medical aid was not sought for four days. The patient was in a state of grave shock and exhaustion when he presented himself for treatment.

Operation. The sac was widely opened and found to contain a large quantity of dark, offensive fluid, exposing a strangulated and gangrenous coil of the terminal ileum, completely severed at the distal constriction, about 5 inches from the ileocecal junction. The wound was enlarged so as to freely expose the right lower abdominal quadrant. Foul-smelling fluid and contents of the bowel had been extravasated and an acute peritonitis was in evidence. An immediate resection of 26 inches of the intestine was made and a lateral anastomosis performed, the Connell technic being employed. The upper part of the abdominal wound was closed, while the lower part, with the scrotal incision, was left wide open and packed loosely with gauze; no radical cure of the hernia had been attempted. The recovery of this remarkable case was uneventful.

Case II.—C. W. G. Male, aged fifty-seven years. Referred to the writer's service in the Ohio Valley General Hospital by Dr. Myron K. Reppard, November 12, 1919. For several years the patient had been subject to mild attacks of indigestion and was conscious of an irreducible lump in his right groin. This lump was manifestly an incarcerated femoral hernia and was so diagnosed by his family physician. There was no other previous morbid personal history. Three days before admission he developed acute tenderness and pain in the right groin, with nausea and persistent vomiting, and he then noticed an appreciable increase in the size of the protrusion. Efforts to move the bowels were fruitless and taxis was of no avail. The pain, which was at first local, became general, and was described by the patient as "doubling up," and rendering him "unconscious."

Physical Examination. The patient was a poorly nourished white man, appearing ten years older than his fifty-seven years. The countenance was drawn and anxious, skin moist, pulse frequent and small, respirations shallow and costal. He lay with his knees drawn up, apparently free from pain, except on palpation of the abdomen. There was entire absence of splenic and hepatic dulness. Over the right saphenous opening there could be felt a protrusion, immovable and semifluctuant, and about the shape and size of an egg. There was absolute stomach intolerance; the ejecta was brownish and foul. The clinical picture was obviously one of strangulated femoral hernia, associated with peritonitis. While this of itself was grave enough from the beginning, it was further complicated by shock which had suddenly developed in the course of an extremely rough trip of seventy miles while in transit from his home to the hospital. The imperative necessity for immediate surgical interference was obvious.

Operation. A preliminary gastric lavage was done. Under nitrous-oxide and oxygen and local blocking an incision over the protrusion was made. This revealed a loop of lusterless, black and lifeless gut, about 10 cm. in length. A foul-smelling, dark-colored fluid, containing fecal matter, escaped from the sac. At the point of constriction one end of the devitalized loop was wide open, and the segment from which it was separated was nowhere to be seen. The constriction was divided and a liberal incision made through the rectus muscle, freely exposing the right iliac fossa. The open and retracted proximal end of the terminal ileum was then identified. It exhibited a sharply defined area of pressure necrosis and an extension of the gangrene about 3 cm. beyond the point of rupture. The adjacent mesentery was thickened, with evidence of clotting in the vessels. The abdominal cavity contained fecal matter, free fluid and gas, and the presenting intestinal coils were studded with a fibropurulent exudate, confined for the most part to the right lower abdominal quadrant. The distal end of the gut, with the gangrenous loop, was withdrawn through the femoral ring.

Fourteen centimeters of the ileum were resected at a point 45 cm. from the ileocecal junction A precautionary mattress stitch was inserted, followed by an end-to-end reunion of the intestinal continuity by the use of a Murphy button. The cut edges of the mesentery were approximated by interrupted stitches of fine chromic gut. After a careful peritoneal toilet and well-directed drainage the abdominal incision was partly closed. The original incision over the hernial protrusion was loosely packed with washed iodoform gauze. Inspection of the resected segment of gut showed two small ulcerations about 1½ cm. in diameter—one within the lumen and a second distal to the constriction. The ulcers were not perforated.

Progress of the Case. The patient responded to measures directed against the shock. The presence of hiccough was an embarrassing feature for twenty-four hours, but it finally yielded to gastric lavage. There was a gradual abatement of all distressing symptoms and a free evacuation of the bowels on the fourth day. The patient's convalescence was uneventful, and he was discharged well from the hospital on the twentieth day. The button was passed on the thirtieth day, and he has continued in good health to the present time.

Mechanical Devices in Anastomosis. In the light of the increasing opposition to the use of mechanical aids in effecting an intestinal reunion, perhaps some apology is due for the selection of the button in this instance.

Thirty years ago Caird,[5] of Scotland, observed that the best results in surgery of the intestine could be gained with needle and thread, and he prophesied that many of the ingenious inventions employed in uniting the bowel would share the fate of the too greatly neglected device of Quatre Maitre, who, in the thirteenth century, employed a calf's trachea for a purpose similar to that which led Ramhdor to use a tallow candle and Senn to employ decalcified bone plates. Certainly, no protest need be made when the gravity of the case demands speed if the Murphy button forms a part of the emergency equipment,

even though we concede with Moynihan that all mechanical devices "have now no more than an historic interest."

Primary Reunion or Artificial Anus. One's first impulse in such an emergency is to make an effort at conservation of the patient's resources and to establish an artificial anus rather than add further to the already exhausted invalid by any tedious sewing. When planning for this less serious procedure it is well to remember that the patient must later be subjected to an operation having a mortality of 27 per cent (Gibson) in the restoration of the continuity of the gut, and that the making of an artificial anus is but "one step on a perilous road to recovery," as Singly has so fittingly put it. On the other hand, it must be remembered that immediate primary union, when properly done, consumes little, if any, more time, whether effected by suture or by a mechanical device. Gibson, in a very complete *résumé*, quoted by Johnson,[22] found in 101 cases in which an artificial anus was formed there were 53 deaths, a mortality of 52.5 per cent. In 226 cases in which primary resection and reunion of the intestine was made at the time, there were 58 deaths, a mortality of only 26 per cent. Further supporting this contention is the interesting fact that the elder Kocher and Mikulicz were ardent champions of primary resection, while Coley's[6] statistics, collected from six of the larger clinics and based on an analysis of 3268 cases, show a mortality of 23 per cent in favor of immediate reunion. However, on last analysis, each case must be determined on its own merits.

Infrequency of Femoral Hernia in the Male. Regarding the infrequency of femoral hernia in the male, in 61,561 males with hernia in the groin, reported by Gibson[2] from Maydl's series, 2362 were femoral, or as 1 to 25. Hoguet,[7] in giving his observations on 2468 hernia operations, found but 1 case of strangulated femoral hernia in the male.

Ulceration, Gangrene or Perforation of the Bowel Beyond the Hernial Constriction. Whipple[8] and his associates attribute the overwhelming depression and death in these cases of obstruction to an excessive breakdown of tissue pro-

tein, due to the absorption into the blood of a toxic proteose. Werelius[9] is inclined to the opinion that it is a liver inefficiency or a cessation of bile secretion which has a direct bearing on the mortality factors. Lynch[10] and Draper[11] and Eisburg[12] contend that the lethal agent is due to an interference with the internal secretory function of the epithelial cells of the gut itself rather than to bacteriotoxic causes, or of biochemical origin similar to parathyroid or other endocrine secretion. Moynihan[13] has written that the mechanical impediment to the onward flow of intestinal contents is not so much the cause of the collapse of these patients as the overloading, distention and the *ulceration* of the gut above the block, together with the absorption of contents whose bacterial virulence is greatly increased. The last thought opens up a new field, as the ulceration beyond or proximal to the constricted loop, to which the distinguished English surgeon refers, goes on to perforation and fatal peritonitis, unless promptly recognized and relieved. The literature contains several timely reminders of this type of hernial complication, and as some of the cases so nearly parallel the author's report, with your indulgence they will be included in this discussion.

Case I.—S. Hida,[14] while operating for a strangulated inguinal hernia, found the bowel in the hernial sac black and devitalized. The compression anemia and gangrene were found to extend 6 cm. above the seat of the constriction, and in this area, above the stricture, there were two perforations and a generalized peritonitis. Thirty-six centimeters of the bowel were excised and anastomosis made by suture. Repair of the hernia was effected by the Bassini method, and the patient made an uninterrupted recovery.

Case II.—Goodhart[15] describes a case in a woman, aged forty-four years, who was operated upon in Guy's Hospital, London, for a femoral hernia, in 1879. Readmitted two years later, because of recurrence of the hernia and persistent vomit-

ing. She was operated upon again, a mass of omentum being removed and the stricture divided. She died twelve days later, the abdomen becoming distended and painful; the temperature was normal until the day before death, then rose to 102° F. The knuckle of bowel involved in the hernia was close to the cecum. At autopsy an ulceration affecting 12 to 13 feet of bowel extended upward from the incarcerated bowel, which was not ulcerated. The ulcers were arranged in clusters, forming more or less circular patches, extending all around the bowel, though more mesenteric than otherwise. The cause of the ulceration could not be determined, as no obstruction to the circulation could be found.

CASE III.—In a review of 1500 inguinal hernia operations at the Massachusetts General Hospital, Boston, Mass., Lincoln Davis[16] notes that there were 8 deaths. One of these was a case of double hernia in which abdominal pain, distention and vomiting developed after the hernia operation. At a second operation on the eighth day perforated ulcers of the colon were found. Necropsy showed no direct connection between the hernia operation and the peritonitis, which was evidently the the result of the perforation of the intestinal ulcers.

CASE IV.—Sellenings[17] reports 2 cases in which perforation above the hernia occurred prior to operation. In 1 case the patient was a woman, with a history of left-sided inguinal hernia of many years' standing, for which a truss had been worn. The hernia became irreducible and very painful, but within a few hours the patient reduced the mass herself. After the reduction she felt severe abdominal pain, chiefly in the region of the umbilicus, vomited several times and was prostrated. The temperature was 101.6° on admission to the hospital; the abdomen was distended, rigid and tender. At operation a round, solitary perforation of the ileum was found, and about 8 to 10 inches distal to the perforation the gut was contused, showing

signs of recent strangulation. The perforation was sutured, but the hernia was not repaired, owing to the patient's condition. The patient vomited frequently during the first few days after operation, but made a good recovery.

CASE V.—Sellening's second case occurred in a man who had a right inguinal hernia for two years and had worn a truss. On the day of admission to the hospital the hernia came down into the scrotum, and was painful and tender. He also had colicky pains in the abdomen, but did not vomit. The abdominal pain became very severe, and he was sent to the hospital during the night; the abdomen was moderately distended, rigid and tender, especially around the umbilicus. The scrotal tumor was painful on pressure, but was reduced by manipulation. Temperature, 102.8° F. At operation a single perforation was found in the intestines similar to the one in the previous case. The gut distal to this lesion showed no gross alteration, indicating that the compression had not been so great as in the first case. The perforation was sutured; the hernia was not repaired; the patient recovered.

CASE VI.—Macnaughton[18] reports a case of left inguinal hernia in which no attempt at reduction was made. The patient was relieved by hot fomentations and the administration of morphin, followed by a small enema, which caused a movement of the bowels. Later the patient became restless and fainted when attempting to sit up; the pulse was weak, the extremities became cold and he died in a few hours. At autopsy an irregular transverse rupture was found in the small intestine in the left lumbar region; for fully 4 feet below this level the ileum, until it passed into the hernial sac, contained blood and mucus. The hernial sac contained nearly 3 feet of healthy small intestine, the cecum with an elongated vermiform appendix, 5 inches of the ascending colon and a considerable portion of the mesentery; there were no signs of strangulation.

Case VII.—Moir[19] reports a case in a man who had had inguinal hernia for six months, for which he wore a truss. On the night prior to admission to the hospital the hernia had come down and could not be as easily reduced as usual. The patient finally succeeded in reducing it, and almost immediately after felt abdominal pain and vomited. When admitted to the hospital the next day the abdominal pain was severe and the patient was in a state of collapse. At operation a perforation in the small intestine was found, and below it a slight constriction of the gut. The perforation was sutured, a careful toilet of the peritoneal cavity made and a drainage tube inserted into the pelvis. The patient did not rally, and died about four hours after the operation.

Case VIII.—J. F. Erdmann[20] reports a case of non-reducible umbilical hernia in a young woman who had a history of abdominal pain, distention and hiccough persisting for about a week. At operation the intestines above the hernia were found to be matted together, and a slough was found at the proximal and the distal end of the jejunum. Ten inches of intestine were excised and suture anastomosis made. The patient made a good recovery.

In presenting this subject, Theodore Kocher[21] suggested the possibility that in strangulated hernia, as in other forms of bowel constriction, the intestinal wall above the lesion may be involved. He believed this was due primarily to venous stasis coming from an interference with the circulation, thus decreasing the resistance of the mucous membrane to the invasion of bacteria and leading to ulceration, or even to perforation.

This ulceration of the mucosa, whatever its cause, seems to present a definite pathologic entity, and we believe that a note of warning should be sounded against any anastomosis being attempted in a case of strangulated hernia with gangrene, without first carefully inspecting the two segments of the gut, with reference to the integrity of the mucous membrane.

The various theories advanced regarding the lethal agent

coming as a sequel of intestinal block, whether it is the ulceration or trauma of the mucosa, suggested by Moynihan, dehydration, proteose intoxication or the invasion of hemolytic bacterial flora, have their supporters, and valid reasons have been presented, based on painstaking research. Whatever the source of the complex biochemical formula, whose venom will produce a clinical picture so grave and a pathology so destructive, we do know that it makes speedily for *exitus lethalis* unless promptly recognized and relieved.

In conclusion it may be mentioned that in dealing with a strangulated hernia, with death of the bowel, there are at least two mortality factors to be considered in addition to the shock occasioned by the intestinal block:

1. The possibility of an intra-abdominal leak, due to a partial or a complete rupture at the point of the constriction from pressure necrosis.

2. There may be further added a definite pathologic entity in the form of an ulceration of the gut leading to a postoperative perforation. This accident has been recorded with increasing frequency, and this fact should lead to a searching interrogation of the mucous membrane immediately adjacent to or beyond the line of anastomosis, in order to forestall a potential leakage.

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ROENTGENOLOGICAL VISUALIZATION OF THE GALL-BLADDER BY THE INTRAVENOUS INJECTION OF TETRABROMPHENOLPHTHALEIN

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In a previous publication[1] we called attention to the fact that the calcium salt of tetrabromphenolphthalein when injected intravenously permits the visualization of the gall-bladder with the roentgen ray. Earlier animal experiments which had been made with the sodium salt resulted in shadows which were not so satisfactory as the ones obtained later with the calcium salt. There were certain objections, however, to the use of the calcium salt, chief of which was that, owing to its relative insolubility, it was necessary to inject a large amount of fluid in order to give the required amount of the substance. In many cases, also, it was found that unpleasant effects were produced, such as dizziness, headache, nausea and vomiting. Chiefly because of a desire to get away from the unpleasant effects of the injection, we have more recently returned to the use of the more soluble sodium salt.[2] We have learned, furthermore, that at least one of the reasons for the unsatisfactory nature of the shadows which were formerly obtained with this salt was due to the fact that in the experimental animals we at first did not appreciate the

[1] Graham, E. A., and Cole, W. H.: Roentgenologic Examination of the Gall-bladder, Jour. Am. Med. Assn., 1924, lxxxii, 613.

[2] A crystalline sodium salt of tetrabromphenolphthalein has been prepared and can now be obtained from the Mallinckrodt Chemical Works, St. Louis.

importance of having the stomach empty at the time of injection. By exercising this simple precaution, we have now found that shadows may be obtained with the relatively soluble sodium salt which are just as satisfactory as those formerly obtained with

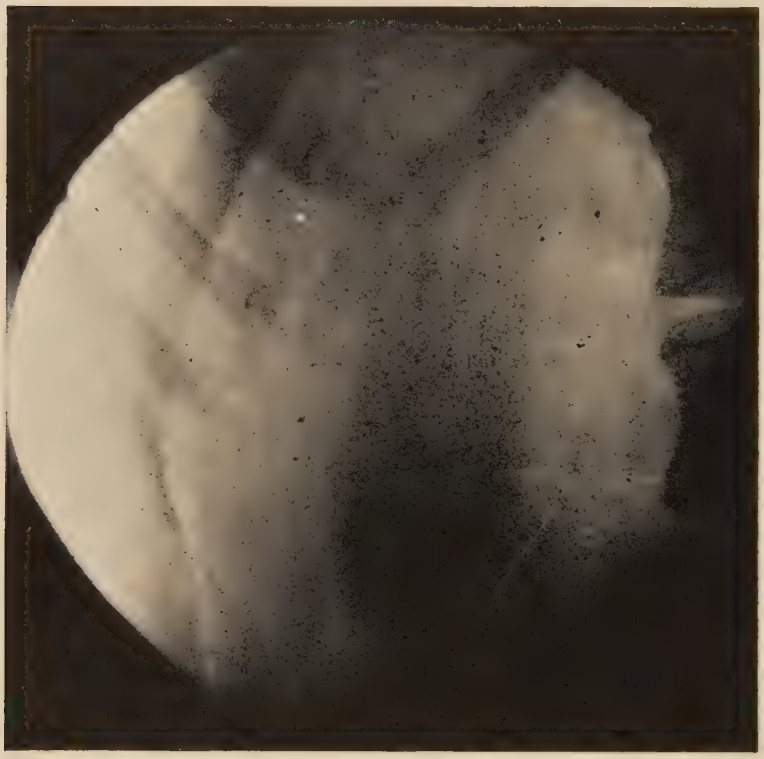


FIG. 1.—The shadow of the gall-bladder appearing faintly seven and a half hours after the injection.

FIGS. 1 to 5.—A normal series.

the less soluble calcium salt. The presence of calcium, therefore, seems to be of no particular value. The additional advantage is also present that instead of the necessity of using about 325 cc of fluid, as was the case with the calcium salt, in order to give the usual dose of 5 or 5.5 gm. of the salt, it is necessary to use

only about 20 or 25 cc of water to effect the solution of the 5 or 5.5 gm. of the sodium salt. The unpleasant symptoms following the injection have also been greatly diminished as a result of the use of the smaller amount of fluid injected. For these reasons, therefore, we are now using the sodium salt exclusively.

FIG. 2.—The same after twenty-four hours. The shadow has become much more distinct and the gall-bladder is much smaller. The contour is regular.

It is hoped that the method of visualization of the gall-bladder, described here, may have a distinct clinical value in the diagnosis of certain cases presenting abnormalities of the biliary tract, which would otherwise be obscure, in a manner analogous to the revolutionary effect of the opaque meal in the diagnosis of

gastrointestinal conditions. Aside from this effect, however, the method may also prove to be of value as a test of the functional capacity of the liver, as judged, for example, by the time required for the appearance of the shadow. It will also permit the addition of new facts to the store of knowledge of the normal

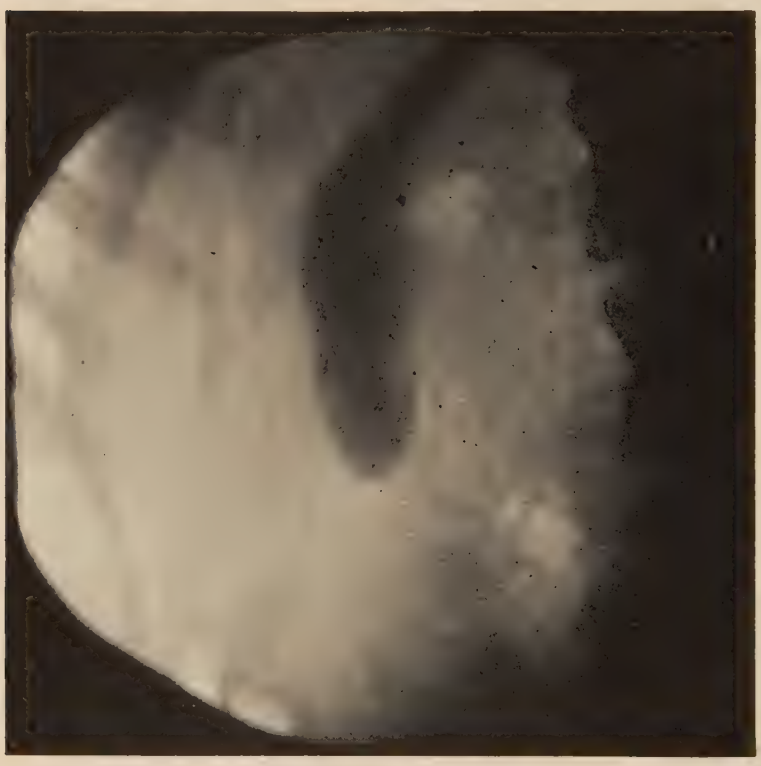


FIG. 3.—The same after twenty-nine hours.

physiology of the gall-bladder, especially as regards the question of emptying time, etc.

The theory of the action of the substance, and the theory upon which the work was based, is that if a substance which is opaque to the roentgen ray would be excreted into the gall-bladder by means of the bile, a shadow should be produced which would

permit an accurate visualization of the gall-bladder. It is necessary, therefore, (1) that the functional capacity of the liver be sufficient to permit it to secrete the substance in the bile; (2) that the cystic duct be open to permit the substance to enter the gall-bladder, and (3) that the concentrating function of the

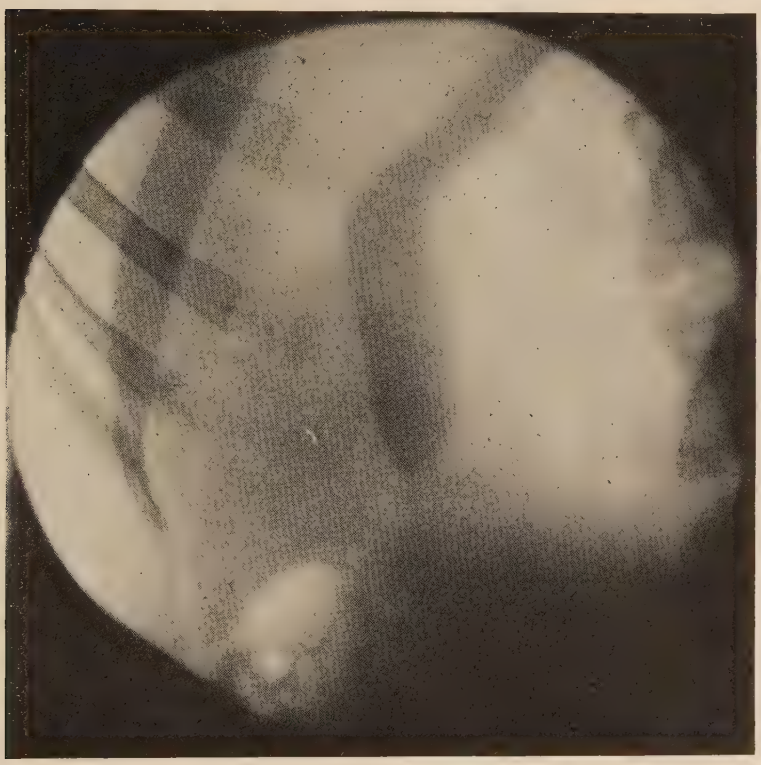


FIG. 4.—After thirty-one hours.

gall-bladder be sufficiently good to permit concentration of the substance in the gall-bladder. Theoretically, therefore, we should expect that the best shadows would be obtained with normal gall-bladders, and that a failure to obtain any shadow at all, after following out the technic given below, would almost certainly denote serious pathology of the biliary tract, including

the gall-bladder. Our experience has borne out the above prophecy.

We have injected either the calcium or the sodium salt now in 54 individual cases, in addition to a large number of experimental animals. In 4 cases we have failed to obtain any shadow, but

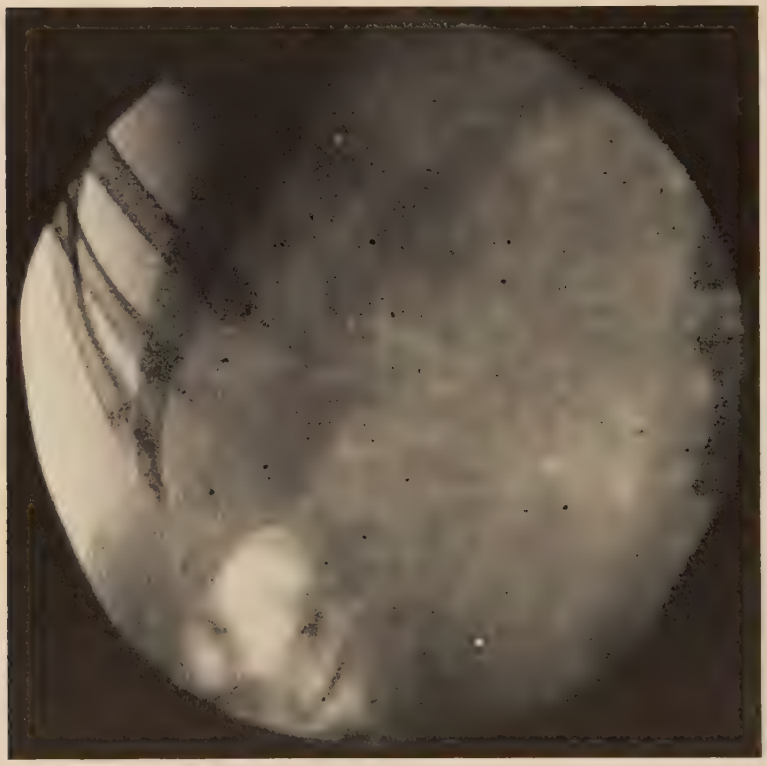


FIG. 5.—After fifty-four hours. The shadow of the gall-bladder has now disappeared.

in all of these, operation revealed very definite cholecystitis. In 2 of the 4 cases there was a complete obstruction of the cystic duct by stone, and in 1 case there was only a fibrous remnant of a gall-bladder associated with a stone in the common duct. We feel therefore, that the failure to obtain a definite shadow

when the test has been carried out properly is of great value, as probably indicating a high-grade cholecystitis. We have also been able to diagnose stones in several instances which have shown themselves as less opaque areas in contrast with the heavier shadow of the rest of the gall-bladder. Adhesions, too, have

FIG. 6.—A gall-bladder, showing distortion by adhesions.

been recognized by distortions of the normal contour of the wall of the gall-bladder.

In order to be sure of the results, it is very essential that a series of plates shall be made, taken at intervals during a period of about thirty-six hours. The interpretation of the normal gall-bladder is based upon the following findings: Usually at

about the fourth to the seventh hour after the injection, a faint but definite outline of the gall-bladder appears which is seen to have the contour of the normally shaped organ, but to be somewhat larger than normal gall-bladders usually seen at a laparotomy. At the end of twenty-four hours the shadow is

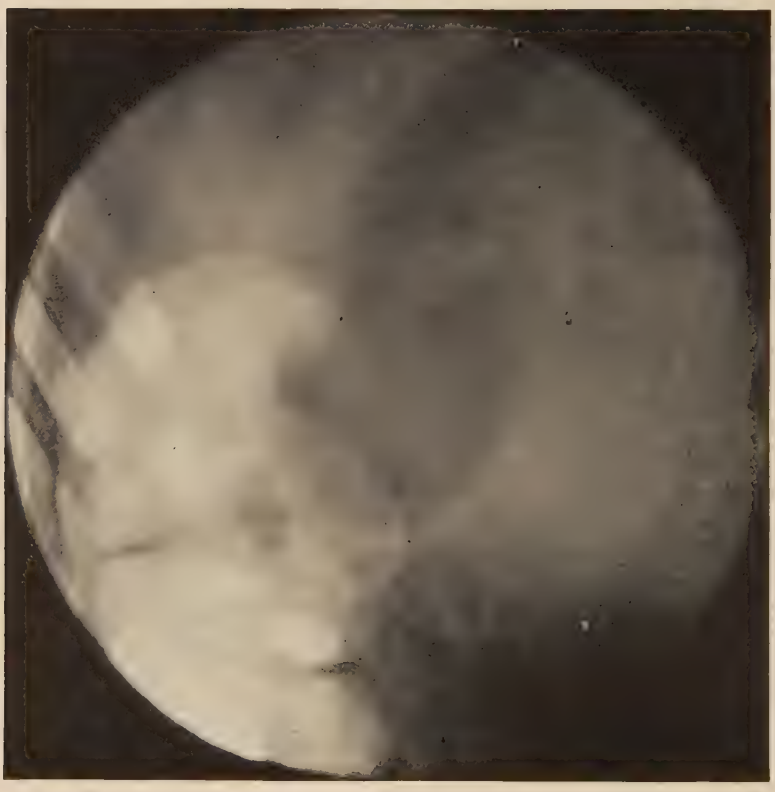


FIG. 7.—A normal gall-bladder, showing through gas in the intestine.

much more distinct, but contracted down to only about one-half of its earlier size. From then on until about the forty-eighth hour the shadow diminishes in size and fades gradually. In cases of simple cholecystitis without stones or adhesions the appearance of the shadow may be delayed, and it may be much

less dense than the normal. At the present time, however, the interpretation of the abnormal shadows is not yet on a secure foundation. A greater experience will be required in order to work out the interpretations satisfactorily.

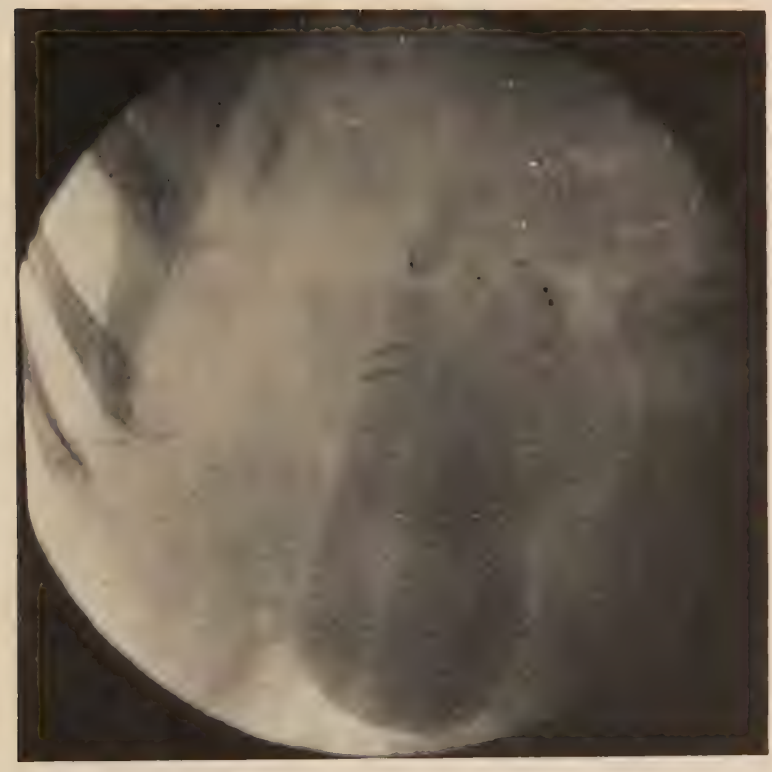


FIG. 8.—A plate which shows a normal gall-bladder superimposed on a kidney shadow.

TECHNIC. Injection of the sodium salt is very easily done with a syringe—preferably in two doses, one-half hour apart. We have not given it all in one dose and do not know if any deleterious results would follow. Much better results are obtained if the injection is made in the morning between 7.30 and 9.30.

The solution is prepared by the addition of 0.8 gm. sodium

hydroxide (16 cc of 3 per cent NaOH) to 5½ gm. of tetrabromphenolphthalein. It dissolves very readily, especially upon heating. After filtering the solution may be sterilized in a boiling-water bath or in an autoclave. If Mallinckrodt's crystalline sodium salt is used the solution is prepared by adding about

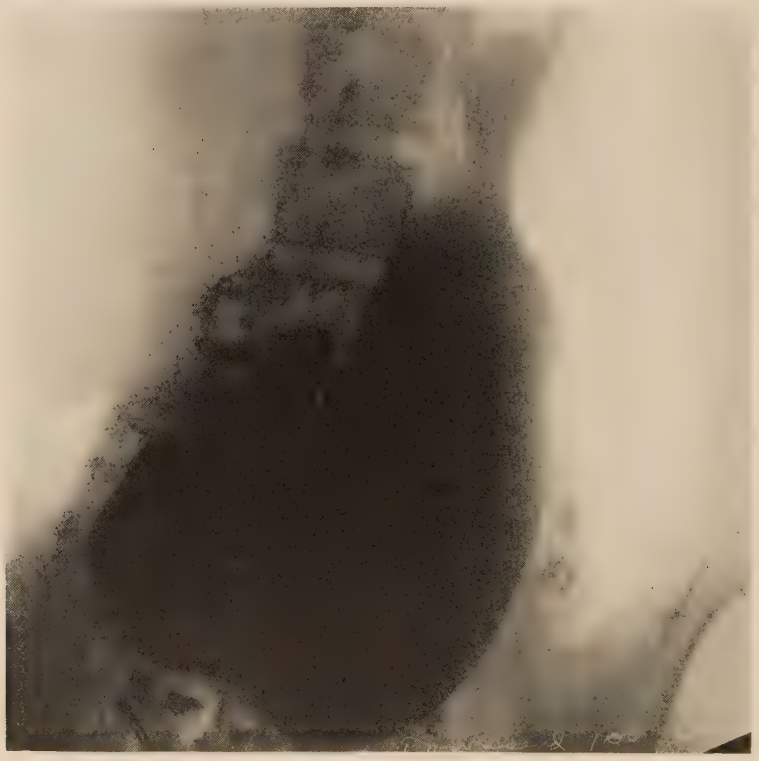


FIG. 9.—A normal gall-bladder shown making a depression on the stomach. The barium in the duodenum is also seen through the gall-bladder shadow.

40 cc distilled water to 5½ gm. of the sodium salt. It is ready for injection after filtration and sterilization. If the patient weighs less than 120 pounds, the dose should be reduced accordingly; great care must be exercised not to allow extravasation of the solution into the arm.

Orders for the patient:

1. Omit breakfast.
2. Omit lunch (may have a glass of milk).
3. Lie on right side of abdomen or be up walking around.
4. Omit proteins from evening meal.
5. May have water by mouth.
6. Sodium bicarbonate, 40 grains (2.5 grammes), every three hours for forty-eight hours.

Roentgenograms are taken at four, eight, twenty-four and thirty-two hours.

The only toxic effects which have, so far, been discovered with the dose recommended have been temporary, and have shown themselves as dizziness, nausea and vomiting. No changes have appeared in the urine. For some unexplained reason, male patients are less susceptible to the toxic effects than female patients. Many patients have no disturbance of any kind.

DISCUSSION

Dr. William L. Estes, Bethlehem, Pa.:

I wish to testify to the value of Dr. Graham's method. Ever since his first publication we have been using it in St. Luke's Hospital and have found it of extreme value, especially in the doubtful cases. In one case recently, which came to operation, the man had been previously operated upon for gall-stone trouble; there was some suspicion of adhesions, and it was a question whether the stomach or gall-bladder was at fault. Operation was undertaken, and the section revealed that the skiagram showed the condition of the gall-bladder quite accurately.

Dr. Allen O. Whipple, New York:

I should like to report a small series of cases in which we have used this method, which was first proposed by Dr. Graham.

In a series of 9 cases we have had 3 patients that showed no shadow. A thirty-six-hour period examination was made in 2 patients, who were subsequently operated upon, and the cystic duct was found completely obstructed. In the third case the duct was almost completely obstructed, and in going over the last film that was taken, we were able, on close examination, to find a very faint shadow.

It has been our experience that the cases that show the sharpest outline in the shortest time are the normal cases. This was impressed upon us particularly in a patient with symptoms that might have been interpreted in a number of ways; the most probable diagnosis we thought was visceroptosis; the shadows appeared very promptly and disappeared very promptly. The intravenous method which Dr. Graham has reported simplifies the procedure, and I feel that, as a diagnostic measure, it will prove a real addition to our present methods. Nothing, of course, will take the place of a well-taken history and a careful examination, but, as we all know, there are a certain number of cases giving indefinite signs in which this may prove of very great value.

Dr. John B. Deaver, Philadelphia, Pa.:

Referring to Dr. Graham's method, I think possibly in an occasional case this may suffice, but it strikes me that this is another instance of machine power overcoming man power. In my experience gall-bladder diagnosis is not difficult; careful palpation and inspection with the proper interpretation of a carefully written history usually suffice to make the diagnosis clear. This is borne out at the operating-table when the pathology has been exposed to sight and touch.

It is a question whether I would not as soon have an operation as to have an injection into my blood system, which must carry more risk than opening the abdomen.

Dr. Howard Lilienthal, New York:

It appears to me that the most important application of this method will be in cases in which a diagnosis of gall-bladder disease is fairly definite. But gall-bladder disease is a chronic condition, and it is easy to be deceived here. One thinks that there is gall-bladder disease, and in many instances "gall-stones" are found in the *x*-ray plates; and then at operation there is found a perfectly normal gall-bladder. This has happened to me two or three times. I think it is in these cases of supposed chronic gall-bladder disease where this method, in perhaps demonstrating a normal gall-bladder, will be of the greatest value.

Dr. Graham (in closing):

I have just a word to say in closing. I agree fully with the implication of Dr. Deaver's remark that there is no machine diagnosis which will take the place of a careful human diagnosis. I do not know what the ultimate value of this will be from the clinical standpoint. I am not even entirely sure at the present time about the interpretation of the deviation from the normal shadows. Only additional experience will bring out these points. Even if it has no value whatever clinically, certainly in some cases it will still serve to clear up a great deal of doubt about the physiology of the gall-bladder. For example, we now know nothing definitely about the normal emptying time of the gall-bladder. It seems to me that this procedure supplies that information. In the second place, if functional liver tests are of any value at all, I think this test will provide not only an *x*-ray diagnosis, but also a functional liver test at the same time.

THE RESULTS OF OPERATIONS FOR CHRONIC APPENDICITIS (SECOND SERIES)

CHARLES L. GIBSON, M.D.
NEW YORK

This paper is supplementary to one published in 1920,[1] which set forth the results of operations for chronic appendicitis in my ward service of the New York Hospital from January, 1913, to July, 1919. In this paper it was pointed out that we had too large a proportion of unsatisfactory results. It was, however, shown that better results were obtained in the last year and that the earlier results fell into a period of reörganization followed by the preparation for and active participation in the World War. The better results began to show so soon as we were able to analyze the causes of our failures and apply better methods.

TABLE I.

Iodine Preparation.

Year	Number of cases.	Per cent unsatisfactory.
1913	70	28
1914	63	23
1915	98	28
1916	100	26
1917	119	24
1918	84	20
Picric Acid Preparation.		
1919	68	13
1920	97	19
1921	105	6
1922	126	7
1923	79	8

[1] Am. Jour. Med. Sci., May, 1920, No. 5, clix, 654.

This second series represents a period of stability and an earnest endeavor to improve our methods. It has been most satisfactory to find that the beginning improvement noted in our first paper has been maintained and improved, and we have only 9 per cent of unsatisfactory, 50 per cent improvement.

Our results are studied by careful attention to the lessons of our follow-up system, which has now been in operation eleven years. This last series shows only 5 per cent without follow-up report, a remarkable showing for a hospital service with many recent immigrants who quickly scatter.

Table II gives a comparison of the results.

TABLE II.—COMPARISON OF THE TWO PERIODS

	July 1, 1913, to July 1, 1919.		July 1, 1919, to January 1, 1924.	
Results:				
1. Excellent	259 (46.0%)	58%	283 (62.0%)	85%
2. Satisfactory	65 (12.0%)		105 (23.0%)	
3. Unsatisfactory	102 (18.0+%)		41 (9.0%)	
4. Unknown	126 (23.0%)		22 (5.0%)	
5. Deaths	3 (0.5%)		3 (0.7%)	
Number of cases	555		454	

NOTE.—In the second series the unsatisfactory results have been cut down 50 per cent.

Our routine comprises a reëxamination by the operator in person after three months. Whatever the result, the patients are told to come back any time they like if they have any complaints, so some of our notes extend over a period of years. Patients with definite complaints (Class C, unsatisfactory) are usually urged to reënter the hospital for examinations, fluoroscopy, cystoscopy, etc. Some of these are relieved by the hospital stay alone—suitable diet, laxatives and suppression of bad liquor, but are still recorded in the unsatisfactory class, although subsequently reporting themselves all right.

To get an accurate classification we have adopted a standard which is in a measure unfair, as it gives an exaggerated value to irrelevant complaints.

Class A. *Excellent* applies only to patients who make absolutely no complaint; pretty hard to attain, as many of these people are naturally a discontented and complaining lot.

Class B. *Satisfactory.* Patient has some complaint, but not directly connected with the operation, *e. g.*, constipation, a normal condition for these sedentary sweatshop workers.

Class C. *Unsatisfactory.* All having definite complaints even if we believe they are liars or neurotics, as many are. For instance, a girl, fat and rosy, who has gained forty pounds, but claims to vomit daily, is included in this class.

In our first paper we formulated the following rules:

"To avoid disappointing results after operations for chronic appendicitis I recommend:

"1. A comprehensive and detailed history.

"2. A complete and thorough physical examination, including all refinements of diagnosis.

"3. Exercise caution in undertaking operation on women as compared to men.

"4. Exercise caution, particularly in the more mature patients, particularly women. In this class other lesions may coexist or may be mistaken for appendicitis.

"5. Avoid the neurasthenics of any age or sex.

"6. Exercise particular restraint when there is no clear and reliable history of well-defined attacks, particularly of localized pain accompanied by nausea or vomiting.

"7. Make a good-sized incision, and, even if a frankly pathological appendix is found, look for other possible lesions.

"8. If no obviously pathological appendix is found, do not cease looking for other lesions until every other possibility has been exhausted; make a supplementary incision if necessary."

From a study of the second series we feel we have nothing to take back in these conclusions, and while essentially no new

knowledge has been acquired, the value of the rules has been emphasized.

Table III gives comparative statistics of some of the main lines of observation.

TABLE III.—COMPARISON OF TWO PERIODS (PER CENT)

	Excellent.		Satisfactory.		Unsatisfactory.		Unknown.	
	1919.	1924.	1919.	1924.	1919.	1924.	1919.	1924.
A. Male	70.0	69.0	14.0	17.0	16.0	9.0	..	5.0
Female	56.0	58.0	16.0	26.0	28.0	11.0	..	5.0
B. History of well-defined attacks	51.0	47.0	51.0	48.0	35.0	18.0	47.0	41.0
C. Kind of incision:								
McBurney	37.0	1.0	22.0	0.0	31.0	0.0	52.0	0.0
Large	63.0	99.0	78.0	100.0	69.0	100.0	48.0	100.0
D. Average number of days spent in hospital	2.57	1.66	2.53	2.07	3.19	2.25	2.30	1.45
E. Special examinations	46.6	17.0	54.0	24.0	56.0	36.0	36.0	18.0
F. Appendix, pathological	85.0	96.0	75.0	95.0	63.0	84.0	77.0	90.0
G. Further exploration	52.0	80.0	83.0	89.0	73.0	84.0	52.0	90.0
H. Subsequent admission	2.0	2.0[1]	0.0	0.9[1]	25.0	32.0	2.0	0.0
I. Subsequent operation	1.5	1.0[1]	0.0	0.0	5.0	4.0	2.0	0.0

[1] Admitted and operated on for conditions in no way related to appendix, *i. e.*, cellulitis of foot. Of the unsatisfactory cases, 32 per cent were males and 68 per cent females. Total number of male cases, 164. Of this number, 14 cases (9 per cent) was unsatisfactory. Total number of female cases, 290. Of this number, 30 cases (11 per cent) were unsatisfactory. Total number of unsatisfactory cases, 44. Of this number, 14 cases were males, (32 per cent); 30 cases were females (68 per cent).

A. It will be noted that women continue to give the most unsatisfactory results. This fact we knew from our earliest studies and tried diligently not to fall into this obvious trap. We feel that the proportion of so-called chronic appendices in women should be diminished to approximate more nearly the ratio of operations for acute appendicitis in the two sexes.

B. The importance of a definite intelligent history of previous attacks. In our latest series of (Class C) unfavorable results only 18 per cent gave this clear-cut picture.

C. Shows the disappearance of the McBurney incision. All our operations permit of thorough exploration.

D. Shows patients were kept longer under observation for the same reason as given in *E.*

E. Shows that more examinations were made in the unsatisfactory (fluorsoscopy, cystoscopy, etc.), these being the cases in which the clear-cut histories were wanting.

F. Bears out the observation of our first series, that in the unsatisfactory cases the appendix is less often recorded in the operative dictation as being distinctly pathological. We have, however, a gratifying increase, 84 per cent, over 63 per cent of our first series.

G. Shows the great increase in the amount of exploration as recorded in the dictation of the operation in our second series—made possible by a proper incision, probably a most important factor in the improved results.

H. Shows that an important percentage of our unsatisfactory results were readmitted for investigation or treatment, but *I* shows that only in a very small per cent were indications for another operation established.

Class C. A comparison of the postoperative complaints (Table IV) shows no essential difference in the two periods. Pain of some kind is the constant complaint. Pain, of course, is hard to check up; we have no means of denying or corroborating the patient's story.

One feature which is disconcerting is the percentage of patients complaining of the "same pain as before operation," 19 per cent for 1919 and 43 per cent for 1924.

The complaints fall into two main groups: Same pain as before operation and gastrointestinal complaints. (Table IV.)

TABLE IV.—POSTOPERATIVE COMPLAINTS OF UNSATISFACTORY CASES.

Same pain as before operation,[1] 18 (4 males, 14 females).
Gastrointestinal complaints, 12 (3 males, 9 females).
Pains everywhere, 2.
Numerous complaints, 2.
Gynecological conditions, 4.
Pain in upper right quadrant, 1.
Pain in upper end of scar and sometimes around the ribs and chest, 1.
Pain over the kidney region, 1.

Class D. Deaths (3).

CASE I.—L. C., aged thirty-four years; male. Appendix pathological. Exploration negative. Developed consolidation at the base of the left lung. Transferred to the medical side. Temperature began to remit and condition of patient became quite satisfactory. On the fourteenth postoperative day suddenly developed dyspnea and complained of a pain in his chest. Died in five minutes. Autopsy showed embolism of pulmonary artery, thrombosis of right internal iliac and infarct of lung.

CASE II.—A. S., aged thirty-four years; male. Appendix not pathological and exploration negative. Convalescence uneventful until ninth postoperative day, when patient got up. Became cyanotic, with great air-hunger, and died in fifteen minutes. Autopsy showed thrombosis in the left internal iliac, with pulmonary embolus at bifurcation of the left pulmonary artery.

CASE III.—H. D., aged thirty-five years; male. Appendix pathological. Exploration negative. On second day following operation the patient began to have acute pain and signs of peritonitis. Revision of first operation. Diffuse peritonitis. Source could not be ascertained. Jejunostomy and drainage. Died shortly after leaving operating-room. No autopsy.

[1] Same pain as before operation—1st series, 1919, 19 per cent.
[2] Same pain as before operation—2d series, 1924, 43 per cent.

Attention was called in our first paper to the coincidence of our beginning better results with the replacement of tincture of iodine by picric acid as a skin disinfectant, introduced in the last half of 1918 and continued to date. We believe it may be a feature, perhaps important of our continued improvement, as it is less irritating and less easily carried into the peritoneal cavity.[1] We note it is coming more into general use.

We believe that unsatisfactory results in surgical practice may be greatly diminished if the will to do better work is established. That a most important factor is the creation of a workable standard on which to base results, a careful analytical study of the causes of poor results and the unflagging desire and search for better methods. If these requirements are fulfilled, better results will more than repay all extra effort.

[1] Gibson, Charles L.: The Advantages of Picric Acid over Tincture of Iodine for Disinfection of the Skin, Annals of Surgery, February, 1919, p. 127; Farr, Charles E.: Picric Acid in Operative Surgery, Annals of Surgery, January, 1921.

ENTEROSTOMY AS A THERAPEUTIC AND DIAGNOSTIC MEASURE

By MILES F. PORTER, M.D.
FORT WAYNE, IND.

Experience and observation have taught me that enterostomy might, with advantage, be resorted to much more often than it is. The term "enterostomy" is here used to signify opening of any part of the intestinal tract regardless as to whether or when closure is to follow. Not as frequently as was the case a few years ago, but still all too frequently, the surgeon is confronted with patients dying from obstruction of the bowel consequent upon an acute abdominal infection, in whom the paramount indication is relief of the abdominal distention and drainage of the intestine, and in whom care of the focus of infection for the time being is of secondary importance, and because of the desperate condition of the patient would better be left to be dealt with later. The following case, reported in brief, is but one of a number occurring in my practice which illustrates the point:

Mrs. A. was seen at her home in the country, at night, *in extremis* from bowel obstruction consequent upon acute puerperal infection. Extreme abdominal distention, leaky skin, vomiting, weak and rapid pulse were the outstanding symptoms. The pelvis was doughy, but there was no fluctuation. A rapid enterostomy done with the patient in her bed was followed by prompt improvement, and three days later the pelvic abscess discharged per vaginam. Complete recovery with closure of the enterostomy followed.

Enterostomy in an obstructed loop of intestine prior to remov-

ing the cause of obstruction will frequently aid in determining whether or not excision is necessary, will lessen the danger of fatal toxemia following relief of the obstruction and make the relief of the obstruction much easier.

Given a volvulus or strangulated hernia wherein the viability of the involved bowel is questionable, opening and draining the gut helps one to determine, more quickly and certainly, for or against excision, and removes the danger of toxemia from the passage of the stagnant content through the remaining part of the gut.

Again, in a case of obstruction without strangulation enterostomy and drainage of the obstructed gut removes the danger of toxemia and facilitates completion of the operation. I have a strong feeling, prompted by considerable experience, that many of the deaths following operation for bowel obstruction might be prevented by opening and draining obstructed bowel of its contents *before the obstruction is removed.*

The danger from soiling the wound may be avoided in large measure, and is not as great as is the danger from absorption of long obstructed bowel content. Summers,[1] of Omaha, and Judd and Rankin,[2] of the Mayo Clinic, have recently published valuable papers on this topic. According to W. J. Mayo,[3] Sir William Taylor "has had noteworthy success in handling acute obstruction of the bowels, using enterostomy for the purpose of emptying the toxic contents of the bowel and of nourishing the patient."

Orr and Haden, in a paper read at the sixty-sixth annual meeting of the Missouri State Medical Association on the "Treatment of Intestinal Obstruction," say: "It is probable that we sometimes do a complete operation when an enterostomy would be the wiser procedure. In the very toxic cases the least operation possible to relieve the obstructed bowel the better. If a bowel will not empty through a good enterostomy opening, it will not

[1] Surg., Gynec. and Obst., 1921, xxxii, 412.
[2] Ibid., 1922, xxxv, 50.
[3] Ibid., 1923, xxxvii, 698.

empty through the natural bowel channel which has been relieved of the obstruction. If an operation for the complete relief of the obstruction, whether it be freeing of a constricted gut, a resection or an anastomosis, is not considered advisable an enterostomy should be done." In previous communications I have cited cases illustrating the difficulty of distinguishing between malignant and benign disease of the hollow viscera even with the abdomen open. In the 1 case reported,[1] the pylorus was the part involved and in the other paper[2] 4 cases are reported wherein non-malignant disease of the colon was mistaken for malignant disease. In 3 of these cases recovery followed colostomy, and in the fourth the growth was excised and an artificial anus made. In this case, after the growth was found to be benign, the artificial anus was closed. In the pyloric case a gastrojejunostomy was done, as a palliative measure, which "palliative" proved curative and the patient reported himself well after four years and eight months. All of these cases were reported by members of this body, 2 by the writer and 3 by men of exceptionally high standing. James C. Masson, of the Mayo Clinic, regards the advice of Telling, Erdman and others to operate in all cases of diverticulitis as "most radical."[3] Of the 289 cases diagnosed in this Clinic only 116 were operated upon.

I have no hesitancy in predicting that in the near future resection for diverticulitis will be rarely done. Evidence is accumulating to prove that many cases of chronic diverticulitis of the sigmoid and rectum, even with tumor formation and symptoms of obstruction, can be cured by colostomy. That colostomy should usually precede resection in those cases demanding this latter operation is, I assume, an established surgical rule. How far the fear of malignancy should lead us in favor of resection as the treatment of choice in certain of these cases is as yet an open question.

[1] International Clinics, vol. iii, 22d series.
[2] New York Medical Journal, February 10, 1912.
[3] Collected Papers, 1921, xiii, 221.

After this paper was finished, I read Horsley's paper,[1] in which he advises enterostomy after resection of the colon. He says, "Patients in whom the colon has been resected make a much smoother convalescence when this enterostomy is done."

Masson says that malignancy and diverticulitis are frequently associated and that the malignancy is no doubt the result of the infection. That colostomy is frequently postponed and in some cases permanently rejected, because of the disagreeable features it entails, to the great detriment of the patient, is not an uncommon experience.

The disagreeableness of an artificial anus has been much exaggerated. Is there a surgeon within hearing who does not know of patients who were subjected to a so-called radical operation for colonic or rectal cancer because of the "disgust and horror" which the thought of colostomy aroused in the mind of the patient or surgeon, or both, when, as the sequence proved, the minor operation offered much more hope for the prolongation of life and promotion of comfort?

Colostomy properly done in properly selected cases adds to the comfort, contentment and pleasure of the patient.

Gant[2] reports that 70 per cent of his colostomized patients have but one movement a day within a month after the operation.

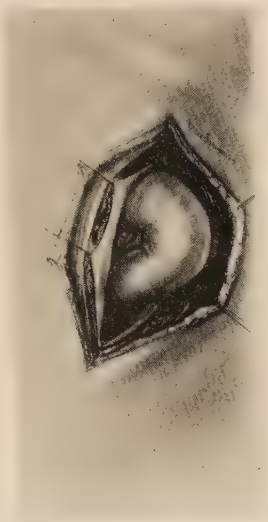
My aim in this paper has been to show that the danger and inconvenience of enterostomy have been exaggerated, while the benefits to be derived therefrom have been underestimated, and that a correct valuation with consequent modification in surgical procedure would be beneficial.

[1] Jour. Am. Med. Assn., April, 1924, p. 1159.
[2] Diseases of the Rectum, Anus and Colon, vol. iii, p. 401.

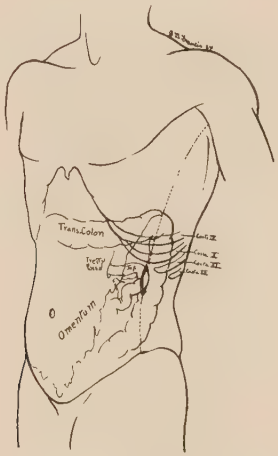
DISCUSSION

Dr. George P. Muller, Philadelphia, Pa.:

Dr. Porter has called attention to the value of enterostomy to an obstruction of the small bowel, and many writers have called



attention to the fact that the higher the obstruction the more toxic the contents. How high one should go in performing this enterostomy is a matter that I will not discuss. In the April issue of the *Annals of Surgery*, in the discussion of the New York Academy of Surgery, the statement is made to "go as high as possible." Sometimes one has



difficulty in deciding the high point of the jejunum; at any rate you are liable to feel portions of the intestine that may perhaps not be stimulated. Dr. Ravdin has worked out for us a technic of tapping the high jejunum which seems to have many advantages, and I have three slides to show you to illustrate this point.

SOME EXPERIENCES IN THE SURGICAL TREATMENT OF CHRONIC VALVULAR DISEASE OF THE HEART

By ELLIOTT C. CUTLER, M.D.
BOSTON, MASS.

(By Invitation)

It is a pleasure to present to you the report of 4 cases of mitral stenosis in which an attempt was made to relieve the mechanical obstruction by means of a surgical method. May I, first of all, however, thank you for your kind invitation and this opportunity? And may I also publicly acknowledge my debt to my associates, Drs. S. A. Levine and C. S. Beck, without whose assistance and stimulation this work could scarcely have succeeded to the point where we can now present it to you?

These experiences are the logical development of an old idea. The suggestion that the stenosis occurring in chronic valvular disease of the heart might be decreased by surgical means was first published by Brunton,[4] of London, in 1902. The papers that appeared immediately following his publication showed that others[27] had considered the possibility earlier, and indeed such a prominent surgeon as Sir Arbuthnot Lane[17] had searched for an operable case. Such an obviously desirable procedure must have received much earlier consideration. Brunton's suggestion, however, fell on deaf ears. The development of intrathoracic surgery was only beginning and the heart was still considered an almost sacred organ. It was only in 1895[5] that the first successful suture of a heart wound had been accomplished; Quenu,[23] Tuffier,[28] Matas[20] and Sauerbruch[26] had just

commenced their differential pressure studies and Meltzer and Auer's[21] method of intratracheal insufflation, which has meant so much in the subsequent development of thoracic surgery, was only introduced seven years later. The surgery of the thorax, however, received a further impetus with Brauer's[3] publication in 1903 of the operation of cardiolysis for adherent pericardium. The suggestions, too, of Forlanini[12] and Murphy[22]

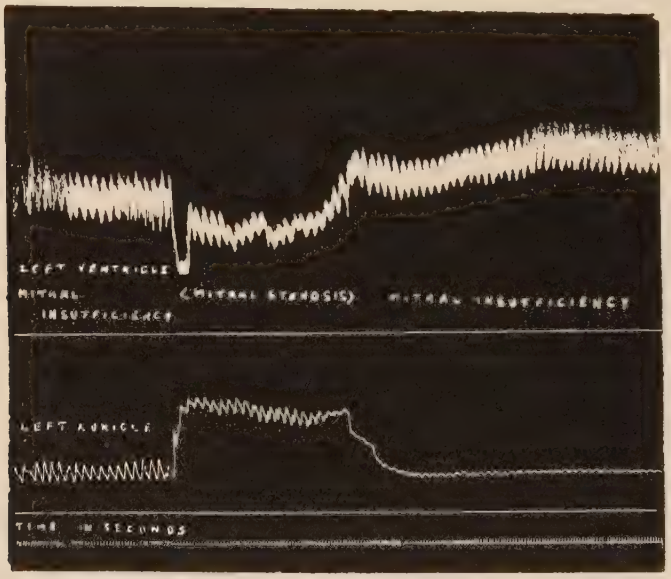


FIG. 1.—Graph recording auricular pressure (above). With narrowing of mitral ring great rise in auricular pressure and fall in ventricular pressure; with release of stenosis return to previous pressures. In this instance a small degree of insufficiency had been established before making the record.

regarding the possibility of pulmonary surgical procedures were a further stimulus.

The most important clinical consideration is what benefits might accrue from relieving a mitral stenosis. It is an accepted fact that insufficiency is more compatible with life than is stenosis. Physiological experiments have shown that with an abruptly created mitral stenosis, either by use of a clamp or a suture at the

mitral ring, there is a most deleterious rise in auricular pressure,[18] and as a result of this a condition of pulmonary stasis that decreases vital capacity and infringes seriously upon the capacity of the blood to sufficiently oxygenate itself.[9] The release of such an experimental stenosis gives a return to normal pressure (Fig. 1). Unfortunately, no method is available for the creation of a permanent stenosis comparable to that seen in human cases.

The fact that pure mechanical mitral stenosis occurs without impairment of the myocardium, and that insufficiency is better tolerated clinically, and relying on the evidence of the benefits that result from the resection of an acute experimental stenosis, operation for the release of such a stenosis seems indicated.

Experimental work in relation to thoracic surgery soon became a favorite field for surgical investigators. It was not long before these men seized upon methods for creating valvular defects used for many years by physiologists. As far back as 1872 Becker[2] had created lesions of the aortic valve by the use of a long glass rod that was inserted into the heart *via* the carotid artery. Quincke had created similar lesions before him, and Klebs,[16] Cohnheim[7] and Rosenbach[24] perfected and enlarged upon these methods. With improvements in surgical technic, and a satisfactory mechanical respiration anesthesia method, these physiological methods were used by surgeons with great success. In 1908 the publication in this country of Cushing and Branch,[8] and in Germany of Haecker,[13] gave the first reports of successful recovery experiments in which the valves were cut by an approach directly through the heart. These methods have been improved upon and enlarged by the work of Carrel and Tuffier[6] and Schepelmann.[25] Recently, Allen and Graham[1] have introduced a new method, using an endoscope carrying both a light and a knife.

These investigations were paralleled by researches into the so-called danger zone of the heart. Thus we know that certain regions in the topography of this organ cannot be used for the insertion of our instruments, viz., the sinoauricular node, the His-Takawara bundle and the region of the coronary vessels.

Elsberg[11] was able to successfully remove the apical one-third of the heart in rabbits, indicating that this area can be safely used for the surgical approach. MacCallum[19] had used the

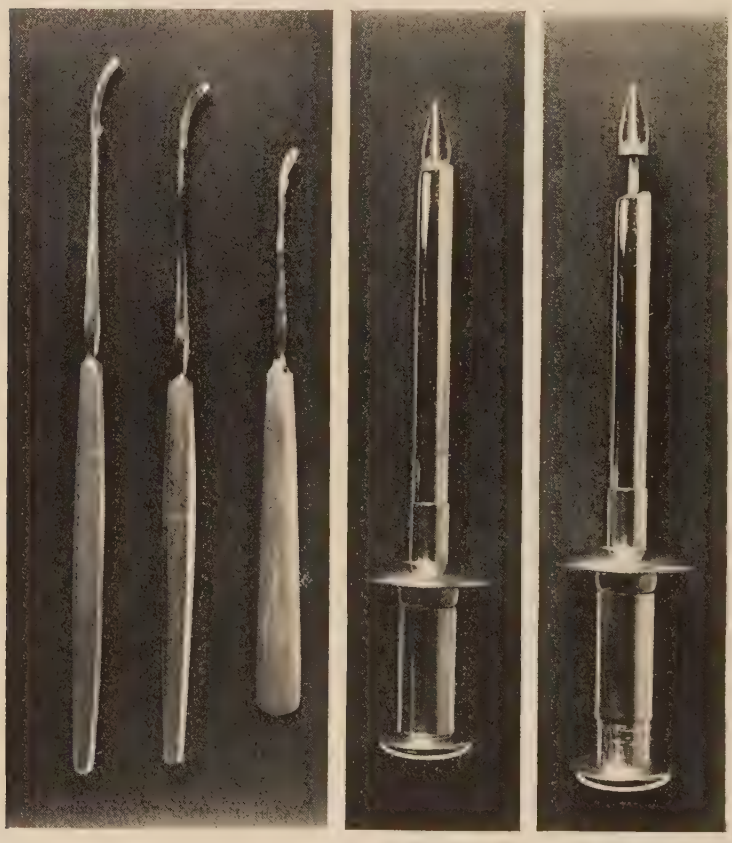


FIG. 2.—Instruments used in cutting valves. The long-handled, small knives were used in the first three cases. The larger instrument (valvulotome) was used in the fourth case.

auricular appendices without disturbing the action of the heart. Thus experimental surgery soon reached a point where valvular insufficiency could be created at will in animals with small risk.

Experiments in the various operative methods previously

proposed for creating valvular insufficiency had been carried out in the laboratory of the Harvard Medical School, when an opportunity to do a human case arrived in May, 1923. At that time we were using a small bladed knife with a long handle and

FIG. 3

FIG. 4

FIGS. 3 and 4.—These represent two actual stenotic mitral orifices. Both small insets represent *x*-ray films demonstrating calcium deposits, those to the left are the valve before cutting and those to the right are of the fragment removed.

approaching through the apical portion of the left ventricle. We had given up the method of approach through the auricular

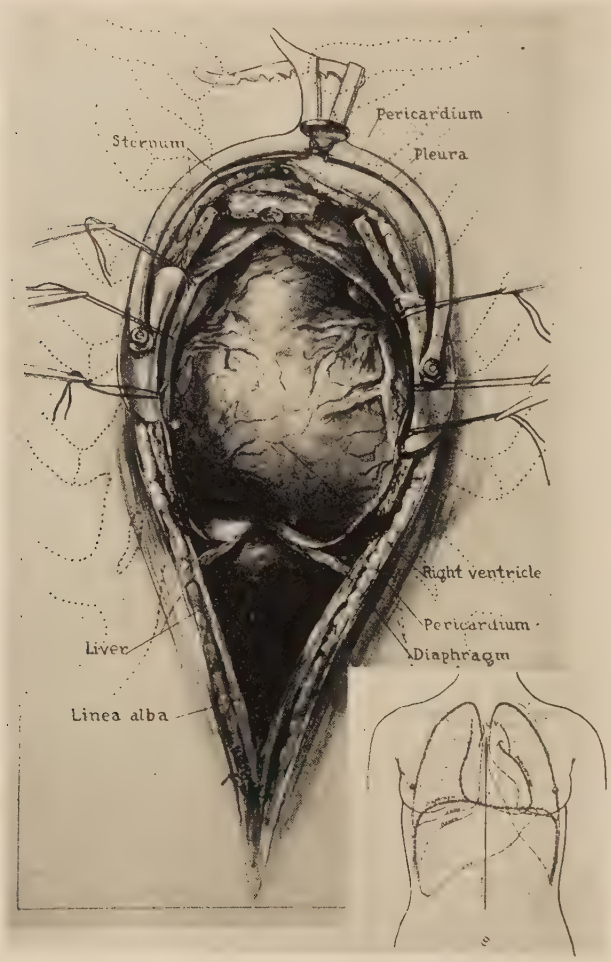


FIG. 5.—Thoraco-abdominal exposure.

appendix as not suitable for use in human cases. Our reasons for this were: (1) The necessity for a transpleural approach, (2) the likelihood of finding a thrombus in the appendix, (3) the

great degree of stasis in the auricle producing the danger of easily tearing it and also of increasing the probability of subsequent thrombosis. We had reached a point where our experience enabled us to create valvular defects by the use of such a small knife inserted *via* the right or left auricles and with small risk to the animal (Fig. 2).

In the first cases we used such a knife, but we early became convinced that such an instrument was inefficient when confronted with the task of cutting the thickened and often calcareous valve leaflets seen in clinical valvular disease. This led to the development of a more powerful instrument (Fig. 2, valvulotome) that utilizes the shearing principle and has the added advantage of actually removing a fragment of the valve. The caliber of the shaft remains the same throughout in order to completely block the opening made in the muscle. Experiments with this instrument on typically stenosed valves in hearts removed at autopsy indicated its change to efficacy (Figs. 3 and 4). Operations upon dogs and goats revealed to our surprise that even such a large instrument caused little disturbance of the action of the heart and created far more satisfactory defective and insufficient valves.

For exposure we have used the Duval-Barasty[10 15] median sternotomy (Fig. 5), that has the advantage of not opening the pleuræ, and which through the manœuver of division of the diaphragm permits wide exposure in the apical region, where it is most necessary. Ether was used for anesthesia in all cases.

Thus far we have operated on 4 cases. One case, the first, is living, well and much improved today, eleven months following the operation. The other cases survived ten hours, twenty hours and six days following the operation.

Case I.—E. L., girl, aged twelve years; born in Boston. Transferred from the House of the Good Samaritan to the Brigham Hospital, May 17, 1923.

History. Good family history. No definite history of chorea or rheumatic fever. Repeated attacks of tonsillitis until ton-

sillectomy six years previous to entry. "Growing pains" at that time.

The present illness commenced three years before admission to the hospital, and consisted first of dyspnea on exertion, later of palpitation and dyspnea, and finally hemoptysis and orthopnea.

Dr. Levine and I saw this patient in the House of the Good Samaritan May 16, 1923, at the request of Dr. Maurice Fremont-Smith, of Boston.

Examination at that time showed an alert child sitting upright in bed with cyanotic lips and rapid though not labored respirations. The chest was prominent. There was a marked thrust in systole and a palpable thrill in diastole. On auscultation one heard a harsh, loud rumble in diastole, ending with a presystolic crescendo. P_2 was accentuated. There was a very faint systolic murmur heard only at the apex. Pulse small, regular and rapid. Considerable number of moist rales constantly at both bases. Blood-pressure, 98/64.

The case was obviously one of advanced mitral stenosis. There was no evidence of a weakened myocardium, and there had not been a febrile attack for some time. Although we would have preferred an older patient, the outlook was so hopeless it seemed justifiable to make the first surgical attempt in this case provided the family would accept the risk after the proposed procedure was explained.

More complete studies in the Brigham Hospital after transfer showed by *x*-ray a slightly enlarged heart with prominence of the left auricle. The electrocardiograph studies indicated an efficient heart muscle with right ventricular preponderance. The vital capacity was 52 per cent of normal.

Operation. May 20, 1923. Cardiotomy and valvulotomy for mitral stenosis. Ether. Median thoracoabdominal sternotomy. Apex suture placed on the heart. One cut made with the small knife. Closure. During operation slight fall in blood-pressure, with recovery after hot saline and adrenalin.

Recovery complicated by pneumonia, right upper lobe, pericarditis with effusion and pain on respiration for three days. Satisfactory recovery thereafter.

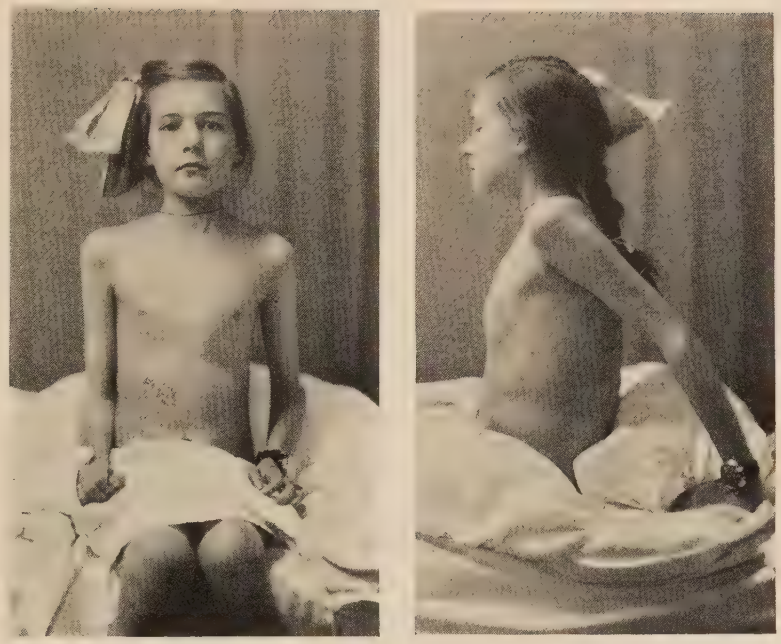


FIG. 6.—Case I. Before operation.

FIG. 7.—Case I. After operation.

At the present time the child is up and about daily in a large family of children, sleeps flat, has no cough or hemoptysis and seems better. Vital capacity is now 60 per cent of normal, and she has gained five pounds.

Present studies show that the heart is about the same size as before operation. The chest bulges in the region of the precordia more than before operation. Blood-pressure, 102/60. Auscultation shows an irregular heart. There is still present a thrill and loud diastolic murmur and also a loud, blowing, systolic murmur. The diastolic murmur loses its intensity as systole is approached. P_2 is greater than A_2, and is accentuated. Subjectively the patient states she feels better than at any time during the last three years (Figs. 6 and 7.)

Case II.—S. G., single woman, aged thirty-five years; born in Russia; dressmaker. Transferred from the Baptist Hospital to the Brigham Hospital, October 4, 1923.

History. Good family history. No heart history; no chorea or rheumatic fever. Attacks of tonsillitis ten years previously; local doctor told her she had a weak heart.

The present illness commenced four years before admission, with palpitation on exertion. Later there followed in order edema of the ankles, weakness and dyspnea. For the last three years the patient was unable to work and was repeatedly in various hospitals.

On October 4, 1923, she was transferred to the Brigham Hospital from the Baptist Hospital, where she had been under the care of Dr. J. H. Pratt, of Boston. There was no division of opinion as to the diagnosis of mitral stenosis. The patient had been observed carefully for over three years, throughout which period she had been constantly fibrillating. The point had been reached when a life in bed was about all that could be looked forward to. She had no family and few friends. She had to support herself. The question of operation with all its risks was proposed to her and gladly accepted, since she realized that the moment she attempted any effort it meant another long period in bed.

At the time of being transferred from the Baptist Hospital she was well nourished and in fair condition. Although thoroughly digitalized, she still complained of palpitation, dyspnea and precordial pain. There was slight cyanosis of the lips and

FIG. 8.—Case II. Auricular and ventricular views of heart with inset of *x*-ray film of valve showing calcareous deposits opening originally slit-like; at upper limits in ventricular view notice sharp edge where knife had cut orifice.

a large liver. Blood-pressure, 116/76. Vital capacity, 66 per cent of normal. Apex rate, 71, absolutely irregular. Moderate systolic murmur and a definite, faint, early, diastolic murmur. P_2 accentuated; no thrill. *X*-ray studies showed increased prominence of the left auricle.

Operation. October 7, 1923. Cardiotomy and valvulotomy for mitral stenosis. Median sternotomy exposure. Ether. Observation of heart most interesting. Huge auricles, both like dark sacs of blood; no contractions whatever except rare convulsive contraction in right auricular appendix. Ventricle small. Three attempts were made to cut the valve. Small knife used. Great resistance was felt. The heart stopped after the last cut, but a little hot saline and adrenalin brought it back, though interestingly enough, with a rate of 50 as compared with one of 165 just previously. Blood-pressures were not readable. Manual massage gave no change. Closure.

Immediate recovery fair. Pulse, 130. Blood-pressure 70 within thirty minutes of completion of operation, and in two hours it was 85. This condition continued for three hours more. The rate then slowed a little, but the blood-pressure continued to fall until imperceptible. Death ten hours after operation.

Autopsy showed no pericardial effusion or reaction. There was a long, thin clot with its point of attachment in the left auricular appendix, from which point it streamed down through the mitral orifice. The mitral orifice was one of those tiny, puckered, stenosed valves with thickened, tough, fibrous edges. At one point the knife had divided the edge, enlarging the opening possibly to twice its original size. Final opening, 12 mm. x 5 mm. (Fig. 8).

Case III.—R. S., single man, aged twenty-six years; born in Massachusetts; no occupation.

History. Good family history. Had rheumatic fever at six years; no history of tonsillitis or chorea, but twenty years ago tonsils and adenoids had been removed at the Boston City Hospital.

The present illness dated back twelve years, when he developed rheumatism again in both knees and was in bed almost continuously for three years. No trouble with any joints afterward. He worked, doing heavy work, up to one and a half years ago, then quite suddenly he developed precordial pain and dyspnea on exertion. He had been unable to work since because of weakness, and remained indoors most of the time. In December, 1923, he was recommended to the hospital.

Examination revealed a pallid, somewhat cyanotic, alert young man; no dyspnea while at rest; pulse regular, rate, 90; blood-pressure, 108/80. The heart was definitely enlarged. Auscultation at the apex revealed loud, snapping first sound, second sound followed by blowing, diastolic murmur, which increased just before the first sound. No systolic murmur. P_2 loud and snapping. A presystolic thrill was felt on palpation. *X*-ray examination showed moderate cardiac enlargement with marked prominence of the left auricle and left pulmonary artery; some pulmonary congestion. Vital capacity, 75 per cent of normal.

The question of operation in this case was thoroughly discussed by the staff and with the family. There seemed little hope that this patient could do much more than he had been doing, sitting around the house. This had become extremely irksome to him and difficult for the family. The latter and the patient were anxious to accept the operation.

Operation. January 12, 1924. Cardiotomy and valvulotomy for mitral stenosis, lysis adherent pericardium. Ether. Median sternotomy exposure. As soon as the heart was exposed, and on opening the pericardium, it was found that anteriorly and inferiorly, and also over a small area posteriorly, there were fairly dense cardiopericardial adhesions. These were separated with scissors and knife until the heart was quite free. Many of the connecting bands were quite tough, but not very vascular, and when all areas had become separated there was no active bleeding, though a slight oozing from some of the cut bands. This manipulation took some time and there was a

steady drop in pressure throughout the procedure. A marked thrill could be felt through the left ventricle much as the sensation derived from feeling a cat when it is purring loudly. A small knife was then inserted through the left ventricle, a definite calcareous mitral orifice was felt, and an attempt made to cut this in two directions at two separate insertions of the knife.

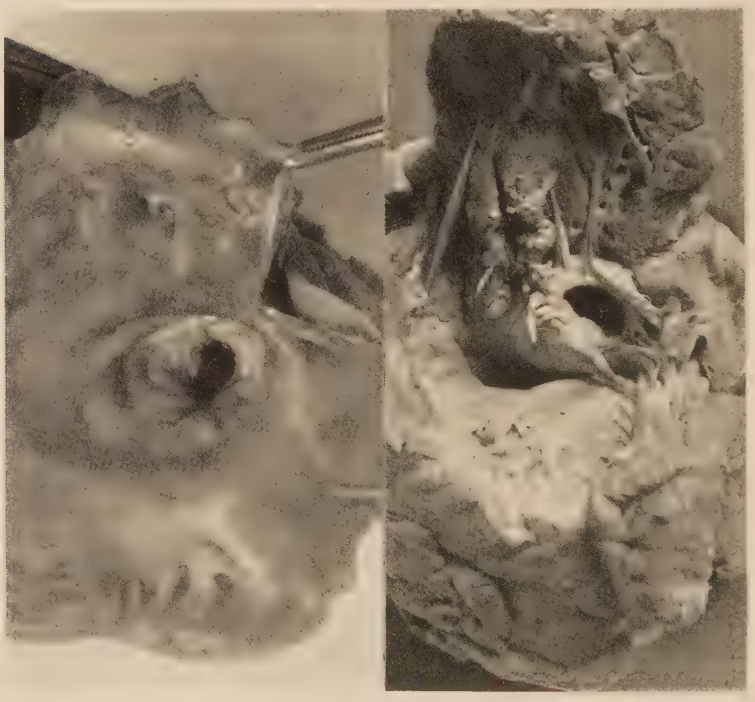


FIG. 9.—Case III. Auricular and ventricular views of heart. Note cuts with knife at both ends of opening and divided chordæ tendinæ.

Heart action continued good; closure as usual; immediate recovery very satisfactory, pressure rising to 95 within an hour and a half.

From this time on the pressure gradually fell. An *x*-ray taken a few hours after operation showed increase in the size of the cardiac shadow. Beginning six hours after operation the

pressure fell steadily and very slowly, in spite of digitalis and caffeine. There was a slight ooze between the skin stitches, which was thought of no consequence. The pulse-rate changed but little. Twenty hours after operation death occurred with signs of failing circulation.

Autopsy revealed a fairly large accumulation of sanguineous fluid in the pericardial sac, about 400 c.c., no clot. Opening the heart revealed a typical calcareous, rumpled, mitral stenotic valve; opening 13 mm. x 4 mm. At the juncture of the aortic and posterolateral cusp the valve had been cut for a distance of only 3 mm. On the opposite edge the cordæ tendinæ had been cut across and there had been some enlargement at the edge of the ring. Probably the orifice had been increased in size one-third by the manipulation. There were no antemortem or postmortem clots (Fig. 9).

Case IV.—M. M., single woman, aged nineteen years; born in Canada, office clerk. Transferred from the Massachusetts General Hospital to the Brigham Hospital, January 28, 1924.

History. Good family history. No definite story of rheumatic fever or chorea. Tonsillectomy as a child.

The present illness dated back three years, commencing first with dyspnea on exertion and only lately precordial pain. Began to have to stay away from work and finally entered the Massachusetts General Hospital, where Dr. Levine and I saw her at the request of Dr. Paul White. There had never been edema or hemoptysis.

Examination showed the thrill and murmur characteristic of mitral stenosis. There was a long, rough, blowing, presystolic murmur extending from diastole to systole. Second sound snapping, P_2+. Electrocardiogram showed right ventricular preponderance and the *x*-ray showed an increase in the shadow in the region of the left auricle. Blood-pressure, 95/55. Lungs clear. Some cyanosis of the lips. Vital capacity, 70 per cent of normal.

The social aspect of the case as a factor in the decision for

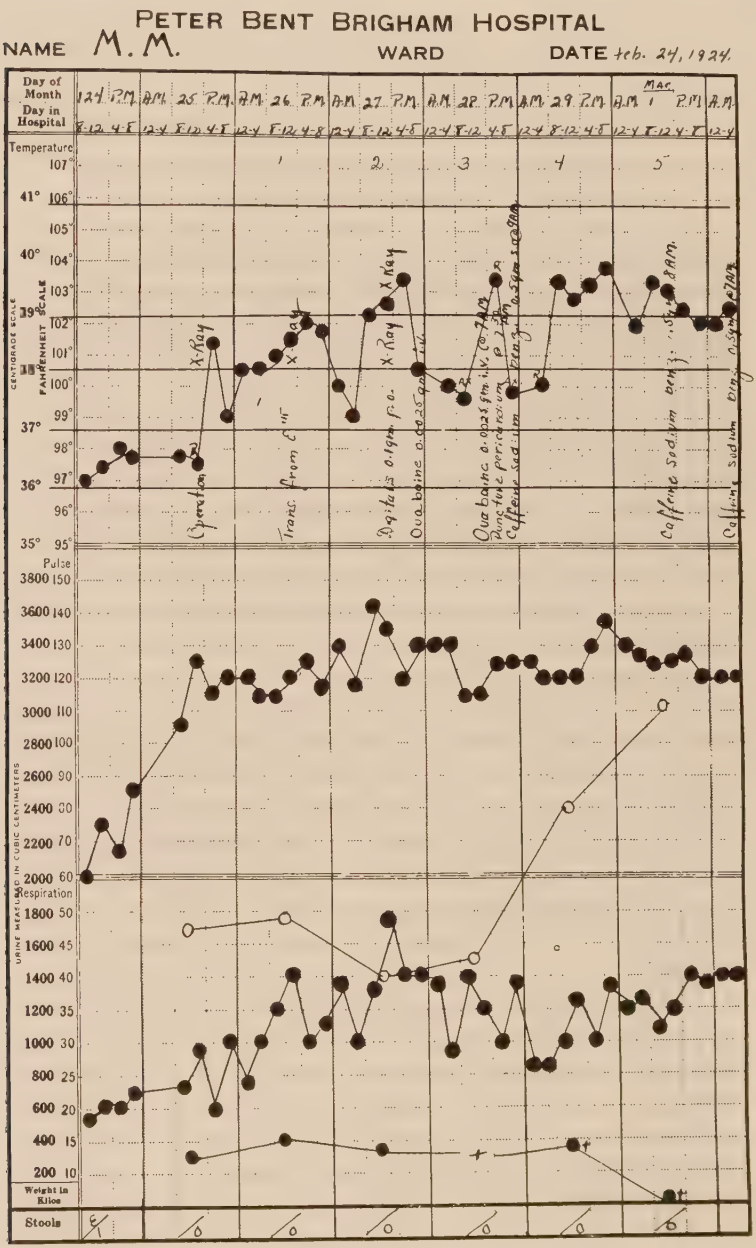


FIG. 10.—Case IV. Clinical chart.

operation. She was dependent on herself for support, and it seemed as though she would be incapacitated from further work. These considerations and the patient's acceptance of the risk after it was fully explained led to our attempting operation.

FIG. 11.—Case IV. Auricular and ventricular views of the heart with insets of piece removed from mitral ring at operation. The tip of the instrument failed to engage the slit-like orifice and was pushed through the base of the aortic cusp, there its cutting edges engaged on the stiff structure when opened and removed a large fragment.

Operation. February 25, 1924. Cardiotomy and partial valvulectomy for mitral stenosis (median sternotomy approach). New valvulotome used. Everything very satisfactory at operation. Pericardium is completely closed.

Good recovery, but temperature, pulse and respirations continued elevated. Blood-pressure came back and remained

around 95 to 100 for first three days. It then fell to 85, but ouabain and caffeine sufficed to bring it back to above 100 for two more days. Pressures then gradually fell to time of death, six days postoperative. Oxygen was used almost contínously Bilateral pneumonia was outspoken as the dominant complication. Repeated *x*-ray studies revealed the heart slowly increasing in size. The friction rub of pericarditis appeared the second day after operation. Pericardial tap on the third day was dry. Repeated examination of the heart sounds and action during the six days postoperative showed absence of the thrill, and all that could be heard of the diastolic rumbling was a very short presystolic murmur. There was also a slight systolic blow at the apex.

Autopsy showed bilateral generalized bronchopneumonia. No increase in pericardial fluid, slight fibrinous pericarditis (traumatic, common in experimental work, pericardium very sensitive). Instrument had created a large defect in the posterior mitral cusp close to the stenosed valve opening. Cause of death, pneumonia (Figs. 10 and 11).

It may not seem to others that we have made much progress, but the experience in each case has greatly enhanced our appreciation of the condition we are to meet and has shown us steps in our technic that were faulty. In the first place, Cases II and III showed us that something more powerful than a simple knife must be used. Case IV proved the efficacy of the new instrument. Case II may possibly be taken as too poor a risk. Case III brings up the problem of the proper procedure in cases complicated by an adherent pericardium.

All the cases have corroborated the deduction from our laboratory work of the relative resistance of the heart to manipulation. We have also benefited from our experience in the use and effect of adrenalin. And the observation in Case II that auricular fibrillation, at least under the conditions imposed in this single instance, may appear to the eye as a complete standstill is of interest. The observation in Cases III and IV demonstrated

beautifully the fact that the thrill of mitral stenosis is produced in the heart and only in that cavity where the lesion exists.

Thus in spite of our discouraging statistics and because no factor has thus far turned up that upsets the sensible proposition underlying this operation, we feel we should continue in our attempts. We should be unworthy of our surgical forbears who passed through an even more disastrous mortality rate in far simpler procedures,[14] did we shirk at this stage shouldering the responsibility of going onward.*

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* Work of this kind can never be represented by a single name. During the first two years the experimental work was carried out with the constant collaboration of Dr. S. A. Levine of the Peter Bent Brigham Hospital staff. In the last year Dr. C. S. Beck has carried on a large share of the investigative work. Their assistance, stimulation and help play an equal share with my own endeavors. The repeated assistance and untiring aid and optimism of Dr. Burgess Gordon, resident physician of the hospital, has helped us over the most trying periods of the final outcome in the three fatal cases. We also wish to acknowledge our indebtedness to Drs. Harvey Cushing and Henry Christian, surgeon-in-chief and physician-in-chief respectively of the Peter Bent Brigham Hospital, whose general attitude has meant more to us than we can ever acknowledge.

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DISCUSSION

Dr. George W. Crile, Cleveland, Ohio:

I wish to congratulate Dr. Cutler on his important contribution to a field in which, heretofore, so little has been done.

Dr. Howard Lilienthal, New York:

I also wish to congratulate Dr. Cutler, as well as his patient, whom I had the pleasure of seeing four weeks after the operation. It was a very striking picture, I can assure you. I think that the profession owes Dr. Cutler a debt of gratitude for demonstrating the possibilities of heart surgery. Much may also be learned from the fatal or unsuccessful cases.

I can only contribute very little, and that is three slides which I have to illustrate the Duval-Barast operation, and while the exposure looks as if it was a very serious matter, it is not shocking. I had one opportunity to test it out in a case of calcified pericardium, the patient, a woman, aged thirty years, who for years was getting progressively worse on account of some obstruction to the inferior cava, which I believe was due in some measure to the pericardial condition. There was no hydrothorax, but there was tremendous ascites, the heart was fibrillating and the patient was in a poor condition as an operative risk, but I hoped that by going in by this method I would succeed in peeling off enough of the calcified pericardium to relieve in part the venous obstruction. I accidentally got into the auricle and had to desist, but in spite of this the patient made an operative recovery and is still living.

The important thing I wish to state is that this operation was not, as was to be expected, followed by shock, but was followed by considerable improvement. There was very slow recurrence of the ascites and the patient did not have to be tapped for six weeks after the operation, although before the operation this was necessary every two weeks.

Dr. Evarts A. Graham, St. Louis, Mo.:

I, too, wish to add my congratulations to Dr. Cutler for this brilliant result which he has obtained and for the additional knowledge which he has given us. I think that surgery of the stenotic heart valves will certainly be a possible procedure as soon as some of the rough places, and the difficulties encountered in learning more about the methods of approach have been overcome.

Dr. Cutler's report of these 4 cases is a great help in overcoming these rough places, which are bound to be encountered in any new type of work.

There are one or two points which I wish to mention supplementary to what Dr. Cutler has said: One, that in 1912 Tuffier forcibly dilated a stenosed aortic valve with his finger, in a young man. In 1920 he reported this case as still being alive and very much improved. His report is in the *Transactions of the International Surgical Society* for 1920. Unfortunately, he does not give us much about the method of approach, and it is impossible to learn exactly where he introduced his finger. At any rate, by the introduction of his finger, he forcibly dilated a stenosed aortic valve, and the man not only survived the operation, but was symptomatically improved.

As many of you know, Dr. Allen and I have been working along somewhat different lines of procedure in these cases for some time, because we thought it was a very desirable thing to be able to see exactly what one was doing. We started on the assumption that any surgical procedure that does not permit vision is not so good as an exact visualization of what you are doing. To permit of this a finer instrument was devised and reported by Dr. Allen, which permits of a direct vision of everything within the heart. Contrary to what one might suppose, it is not impossible to see in the heart with an electrically lighted instrument in spite of the presence of the opaque blood. One can see just as clearly through this instrument as one can see through a cystoscope in the bladder, provided that the lens is put against the object which one wishes to see. It is the same principle as putting the finger in a glass of milk. If you put the finger against the inner edge of the glass it can be seen perfectly.

Another point of disagreement perhaps between Dr. Cutler and ourselves is concerning the method. We feel that the approach through the auricular appendage is much more desirable than one through the ventricle. Although it is quite true that the ventricle has been cut by stab wounds, etc., and it has been sutured successfully, there is a comparatively much narrower margin of safety in the ventricle than in the auricular appendage. You cannot cut the ventricle indiscriminately without great danger to the heart. In order to overcome this, we thought it desirable to cut through the auricular appendage, which is a vestigial structure. You can do anything to it, even amputation, without having any effect whatever on the heart, and, therefore, it seemed a better method of approach with much less danger.

By means of the instrument which Dr. Allen has devised it has been

possible, in a large series of experiments upon dogs (some 50 or 60), to make a cut in the mitral valve of any desired size or location, and it is also possible to remove a wedge if desirable. One can see, therefore, at any time what one is doing. He can note the effect of his procedure, and if that is not enough, he can cut a little more, and he will go through a part of the heart which is relatively immune to any serious effects itself.

Unfortunately, I cannot report any brilliant successes in the human so far with this method. We have had only one opportunity in which to try it, and unfortunately, when we got in we found an abnormal auricle such as we find at autopsy in about 10 per cent of hearts. This abnormality prevented the introduction of the instrument. We were therefore unable to proceed with the section of the mitral valve.

I feel that Dr. Cutler is to be greatly congratulated on having got away successfully with 1 case. Even if it is only 1 out of 10 or 15, it is a great achievement, because any radical new surgical procedure is accompanied by a high mortality, and we must remember that these cases are practically hopeless any way.

There is one question I should like to ask, and that is, How much good the decompression of this large heart had to do with the amelioration of symptoms in this first patient? You will remember that there was considerable bulging of the precordial area. These hearts are large. Possibly a decompressive operation alone would greatly improve the patient.

Dr. W. W. Keen, Philadelphia, Pa.:

I feel that Dr. Cutler is to be encouraged and congratulated on his courage. I thank God I have lived to see this domain in which surgery had not yet entered, entered now definitely and positively! I have often wondered what would have been the fate of anesthesia if Gilbert Abbott, on October 16, 1846, in the Massachusetts General Hospital, had died. I know what was current in Berlin; that when Henry B. Sands, who, as some of the older men here remember, was one of the eminent surgeons in his day in New York, was speaking with von Langenbeck and urging ether on account of its comparative safety, von Langenbeck said to him, "Won't you give ether for me tomorrow morning in my clinic?" Sands etherized the patient, and he died on the table before the knife had touched him. This put back the use of ether for years in Germany.

I think that Dr. Cutler is to be congratulated on the fact that 25 per cent thus far have recovered. I venture to say, if I live long enough, we shall see 50 per cent recover.

Dr. Rudolph Matas, New Orleans, La.:

I cannot resist the impulse to add a few words to the general applause which has so justly greeted Dr. Cutler's splendid achievement, and though these may appear superfluous after the eloquent tribute paid him by our distinguished Fellow, Dr. Keen, and the gentlemen who followed him, I feel, as a fellow-worker, deeply interested for many years in the progress of cardiovascular surgery, that I cannot remain silent in the presence of an event that marks so notable an advance in the history of cardiac surgery. It is only right and proper that those of us who have lived through the pioneer period of cardiac surgery should appreciate the evolutionary significance of the clinical and experimental accomplishments of Drs. Cutler, Allen and Graham, and in congratulating them upon their success, give them our wholehearted support and encouragement in the further prosecution of their splendid work.

This is a memorable meeting in many ways, but in none more striking and delightful than in the assurance given by the contributions of our younger Fellows that the future of this Association and of American surgery is safe in their hands.

Dr. Cutler's successful operation is the actual realization in the living human subject of a dream long ago visualized by Sir Lauder Brunton. The feasibility of valvulotomy in mitral and other cardio-valvular stenotic lesions has been demonstrated in the lower animals by a number of laboratory workers.

The fact that Dr. Cutler's work has been conducted in an institution directed by Dr. Cushing, reminds us of the successful valvulotomies that he (Dr. Cushing) repeatedly performed, in earlier years, as class demonstrations in the artificial production of valuvular lesions while working with Dr. MacCallum, as a teacher of experimental pathology, in the Hunterian Laboratory of the Johns Hopkins Hospital. The later experiments of Carrel, Tuffier and others abundantly confirmed the practicability of this difficult phase of cardiac surgery and laid the foundation for their clinical application. No one, however, had the courage or temerity to deliberately attack the problem as it presents itself at the bedside, until Cutler demonstrated that the feat could be accomplished in the pathological heart with all the success and precision of a purely physiological experiment. There is, indeed, a great leap between the successful suture of a heart wound or the extraction of an intracardial projectile—which until now has been regarded as a supreme achievement in cardiac surgery—and the carefully planned valvulotomies for stenotic-mitral disease which Dr. Cutler has so brilliantly performed and described today. The

methods of approach and the instrumentation devised by Cutler and by Allen and Graham have revolutionized our preconceived notions and brought the operation within the bounds of legitimate surgery.

We should not forget, however, that these operations are suggested and practised only for the relief of advanced valvular lesions, chiefly mitral, and that it is precisely in this class of cases that the patients are the least fit to undergo any serious surgical intervention. It adds no little to Dr. Cutler's merit that he should have succeeded in spite of these usually forbidding conditions.

As a whole, the outlook of cardiac surgery in dealing with the new methods of approach and instrumentation devised by Dr. Cutler and by Drs. Allen and Graham, valvular cardiopathies of an obstructive type may be attacked at an earlier stage and with greater safety than would seem justified at present, and without the grave doubts and anxieties that are inseparable from all operations performed in the advanced and terminal stages of heart disease. Therefore, without being oversanguine in our expectations and fully realizing the limitations of this phase of surgery, we must rejoice at the larger and more encouraging vision of the future of intracardiac surgery that is vouchsafed us by the epochal contributions of Drs. Cutler and Allen and their associates.

Dr. Harvey Cushing, Boston, Mass.:

One feature of this study which has not been emphasized in the discussion is its Hunterian characteristics. Dr. Cutler has given us, in a brief fifteen minutes, the report of a series of four operations in which for the first time an operation for mitral stenosis has been undertaken. There is nothing new in the idea, for the possibility that the surgical transference of a mitral stenosis into an insufficiency might be beneficial to the patient was suggested many years ago by Sir Lauder Brunton.

What will be the outcome of these and similar operations time only can tell, but what I wish to particularly call attention to is the long and detailed series of experimental observations which were antecedent to these operations. Studies such as those which have been made by Dr. Graham and by Dr. Cutler and their co-workers represent years of painstaking experimentation on animals, to which many others in years gone by have contributed their bit, as the reader of this paper would have made clear had time permitted.

In Dr. Cutler's case, the opportunity and the man have happened to meet. In our felicitations to him on his courage and skill it must not be forgotten that he first had to secure the confidence of physi-

cians who likewise shared in the responsibility. Their confidence and support were gained by a knowledge of the laboratory studies which had gone before and had given the experience which alone justified the operations he has described. It adds one more telling argument in favor of animal experimentation which, properly conducted, is something the profession must continue to support in spite of those ill-advised people calling themselves antivivisectionists, who vigorously oppose the use of animals for these purposes.

Dr. Crile:

I would like to emphasize what Dr. Cushing has said. This together with many of the other great advances which have been made in surgery has been based entirely upon experimental researches, and I heartily agree that we all ought to support, and on all occasions to maintain, our right to employ animal experimentation in order to solve the important problems in surgery. It would hardly be possible to suppose that this advance, as well as other great advances made in the last few years, could have been made without the aid of experimental medicine.

Dr. Cutler (in closing):

I should like first to thank Dr. Keen not only for what he has said today, but for what his letters have meant to us in the past two or three months. Although I had never met Dr. Keen, he was interested and kind enough to write us many letters, which have always been a great help. When we were discouraged because of our mortality rate, he encouraged us and pointed out that in the early days of surgery the mortality rates were often high in far simpler procedures.

Dr. Lilienthal has brought up the question of the exposure, and I should like to emphasize what he has stated in regard to the simplicity and amplitude of the procedure used. Although this exposure appears as a very elaborate undertaking, it is not a shocking procedure. In the first place, it is in the midline where there are no important nerves and no great bloodvessels, and although the incision is long, we have a minimum of trauma. Even in such poor risk cases as the ones reported, there was practically no shock following the procedure.

Dr. Cushing's and Dr. Crile's remarks, which they have just made about the value of animal experimentation, emphasize an important point. We would never have dared to operate on a human case without an extensive experience in animal investigations beforehand. We had to learn how to handle the heart and how much trauma it would stand, in addition to developing a satisfactory instrument.

I would like to refer to the case of Prof. Tuffier, mentioned by Dr. Graham. After corresponding with Dr. Tuffier, I do not think he feels the case was greatly improved, and it is difficult to imagine how digital dilatation of a stenotic orifice could accomplish much. The pain disappeared and did not return. He also stated in his address before the International Surgical Congress in 1920 that Doyen had attempted to operate on a case of mitral stenosis previously, but that an interventricular defect was found at operation. The patient did not recover. I wrote to get more data, but he could not give me the reference to Doyen's paper.

The use of the cardioscope brings up a very interesting point: Our interest in heart surgery dates back to 1913, when Lawrence Rhea and I. C. Walker, working in the laboratories of the department of medicine at Harvard, spent a year in trying to do this same thing. They had made for them a cardioscope which we also used without success. It had a knife and carried a light and a lens at the tip, but the lens was not rounded. We were not satisfied with what we could see or do with this instrument. We also feel that the auricular approach carries too great a risk. The question of approaching through the auricle or the ventricle is of much interest. The auricular appendix approach had been used previously by Mac Callum and by Rosenbach. The difficulties with this approach are: First, that the appendix is much smaller relatively in human beings than it is in dogs, and, second, there is a dilated and thinned auricle in cases of mitral stenosis. In fact, it is a serious question whether one would even dare to invaginate it. I should be very much afraid of it tearing. In such cases, where it is very thin, there would also be stagnant blood, and with this condition the danger of thrombosis increases. There may even be old thrombi in the auricular appendix or even an obliterated appendix in human cases of mitral stenosis. If this is present this route fails of use at the critical moment. The thrombus formation would also be a real threat after operation, because of stasis of the blood stream and injury to the intima. I may be quite wrong, but I do not believe it is as safe as going through the ventricle. However, we are not discouraged with the principle of the cardioscope, but it must be incorporated with a powerful instrument, so constructed as to actually remove a fragment from the stenotic ring.

ESOPHAGOTOMY FOR FOREIGN BODY

By P. E. TRUESDALE, M.D.
FALL RIVER, MASS.

Foreign bodies arrested in the esophagus command attention because their presence is a source of great danger and their removal becomes immediately a very vital problem.

Children often fall victims to this accident because they are in the habit of placing a great variety of articles in their mouths. In adults, about 62 per cent of the cases are due to chicken bones, fish bones or poorly fitted artificial teeth.

In regard to the situation at which foreign bodies may become lodged, it has been observed that small, pointed bodies which easily penetrate the mucous membrane may become fixed at any point in the esophagus. Very large bodies usually cannot pass the isthmus and remain fast in the pharynx. The larger variety of foreign body which has passed through the pharynx most frequently lodges at those places where, under normal conditions, the esophagus is constricted: (1) just behind the cricoid cartilage, (2) the middle constriction which is about opposite the bifurcation of the trachea and on a level with the 7th cervical vertebra, and (3) the inferior constriction where the esophagus passes through the diaphragm. Foreign bodies which reach the inferior constriction and stop there usually have been forced down by attempts to get them into the stomach with bougies. The spontaneous descent of larger foreign bodies to the lowest constriction is rare. The majority of foreign bodies remain lodged in the cervical portion of the esophagus. Kronlein explains this by the fact that here the esophagus is wedged in between the vertebral column, the larynx, the thyroid cartilage and in the aperture of the thorax.

Among the earliest writers upon this subject the French appear to have contributed the most valuable monograms. Although Goursald[1] was the first to perform esophagotomy for this condition in 1738, Guattani[2] was the earliest systematic writer on the technic of the operation. Sir William Ferguson, in his surgery, published a valuable treatise, in which he cautioned his readers that however simple the operation seemed on the dead subject, it was attended with much labor on the living, and the surgeon could escape its hazards only by the most painstaking dissection. Among American surgeons the contributions of Dr. David Cheever, of Boston, in a monogram published in 1868, were notable. He made an exhaustive review of the literature on the subject, and reported 3 cases operated upon by himself.

He emphasized the fact that attempts at extraction by mouth were capable of infinite mischief; that owing to the structure and connections of the esophagus, its walls are especially prone to perforation, and such accidents have occurred from the use of the probang. The esophagus is united to the prevertebral muscles only by loose connective tissue. On this account it is readily pushed before the probe and injured. In this respect it is analogous to the membranous portion of the urethra and should be handled with meticulous care. Deaths from efforts to remove foreign bodies in the esophagus through the mouth are more common than is generally supposed.

In Keen's *Surgery* will be found a most excellent chapter dealing with this subject. Ingalls,[3] writing in 1912, said, 'A few years ago the removal of foreign bodies in the esophagus by means of the esophagoscope was considered devoid of danger, but we know that fatalities may occur, and we have reason to believe that in general there may be a large percentage of mortality."

Among the modern authorities the work of Chevalier Jackson,[4] of Philadelphia, stands out preëminently. He has perfected the

[1] Cited by Guattani, loc. cit.
[2] Memoirs de l'Acadèmie de Chirurgie, 1819, Tome i.
[3] Am. Jour. Surg., January, 1912, p. 42.
[4] Bronchoscopy and Esophagoscopy.

operative technic through the esophagoscope to a very high degree and thus placed a sharp limit on the cases needing the open operation. His mortality of 2 per cent or less represents the most skilful work among trained operators. Jackson feels that the mortality in the hands of those without adequate training and experience must be many times greater than this. He regards the esophagoscope in the hands of rough, careless and unskilled physicians as a dangerous and often fatal instrument. Moreover, there are risks associated with the use of the esophagoscope which he describes as "complications and dangers." Asphyxia from pressure of the foreign body or the foreign body plus the esophagoscope is a possibility; faulty position of the patient, with faulty direction of the esophagoscope, may cause alarming symptoms from pressure upon the trachea, especially when the patient is under general anesthesia. Prompt introduction of a bronchoscope with oxygen and amyl nitrite insufflation and artificial respiration may be necessary to save life. Whenever cocain is used the danger of poisoning cannot be ignored. Perforation of the esophagus with the esophagoscope, while rare in skilful hands, is a recognized liability, especially when the esophageal wall is weakened by ulceration or trauma. In the presence of these pathological changes there exists a danger of making a false passage or entering one with the esophagoscope. At the cricopharyngeal constriction fatal esophagoscopic perforation by inexperienced operators is very likely to occur. Here there is a weakly supported area in the esophageal wall. Richardson[1] refers to the liability of impaction of instruments used by mouth, thus requiring esophagotomy for their release. Such are the dangers observed in every-day practice and avoided only by expert knowledge and perfected technic.

However, in spite of these discordant facts, endoscopic procedures for the extraction of foreign bodies in the esophagus are generally considered preferable to and safer than the operation of esophagotomy, providing the esophagoscopist possesses adequate skill and equipment. Nevertheless, a method known to be

[1] Dennis: System of Surgery, iv, 233.

safe in the hands of one possessing the necessary attainments may prove an unhappy adventure for another ill qualified by lack of training and experience. The most able exponents of esophagoscopy invariably refer to "the skill of the operator" as the *sine qua non* for success. By this reference I am sure that just ordinary skill is not implied. It is not to be construed as that degree of skill possessed by the average capable laryngologist. Even the moderately difficult cases demand talent, ingenuity, adroitness, perhaps consummate skill acquired only after many years of convergent practice.

In his chapter on Acquiring Skill, Jackson remarks: "Endoscopic skill cannot be bought with the instruments. Repeated exercise of a particular series of maneuvers is necessary. As with learning to play a musical instrument, a fundamental knowledge of technic, positions and landmarks is necessary, after which only continued manual practice makes for proficiency. Endoscopy is a purely manual procedure, hence to know how is not enough—manual practice is necessary. Practice on the cadaver, on the rubber-tube manikin and finally upon dogs should be pursued for the education of the eye and the fingers. It is inhuman and impossible to obtain the preliminary experience on the living subject."

Providing a patient must receive prompt relief and is located in a district remote from an endoscopic clinic, the method of dealing with the situation is of prime importance, because it is likely to make all the difference between a triumph and a fatal failure. No doubt more of these cases can wait and be transported long distances than the exigencies of the situations usually appear to warrant. Notwithstanding this fact, the picture presented by a patient strangling, choking and partially stifled by a large foreign body arrested in the esophagus is sufficient cause for infirmity of purpose, fright and even panic on the part of his attendants. Four such cases have occurred in our community. To my knowledge three died after the use of bloodless methods, at first presumably conservative, then, as in 2 cases, followed by esophagotomy.

The fourth case, a male, aged twenty-three years, was brought to our hospital from Warren, R. I., on September 6, 1923, two hours after he had accidentally swallowed a partial denture of the upper jaw. No attempts had been made to dislodge the plate except by the patient himself. He tossed about ceaselessly in efforts to move the foreign body up or down. Upon examination his appearance showed evidence of exhaustion and prostration. He was dyspneic and salivated freely. There was moderate swelling of the neck and marked tenderness at the suprasternal notch. Otherwise his physical condition was good.

Pain was severe and persistent, requiring hypodermic injections of morphin. The roentgen film (Fig. 1) showed a dental plate in the esophagus arrested opposite the 7th cervical vertebra and about at the aperture of the thorax.

A conference was held among members of the hospital staff and by telephonic communications with Dr. Chevalier Jackson, of Philadelphia, and Dr. Crosbie Greene, of Boston, after which we reached the conclusion that under the circumstances the open operation here offered this patient the best chance for recovery. Our decision was based upon the following premises: (*a*) The foreign body was large, horseshoe in shape, with rough edges, and impacted in the cervical portion of the esophagus. (*b*) It was definitely localized and accessible by direct approach. (*c*) The patient was dyspneic and weary. He could not swallow water without convulsive effort, causing an aggravation of pain and soreness. (*d*) He was a young, healthy adult with a long, lean neck and no enlargement of the thyroid gland. (*e*) No trial procedures had been undertaken, hence no complications had been superimposed. (*f*) Available for the operative effort were a laryngologist and a general surgeon. The former was very capable of doing those operations usual in the practice of laryngology, but lacked familiarity with the use of the esophagoscope. (*g*) There was neither equipment nor trained assistants with which to venture an endoscopic procedure.

Dr. Crosbie Greene recommended esophagotomy. Dr. Chevalier Jackson believed that the denture could be removed

by mouth, but that local conditions should help determine the plan of operation.

Inasmuch as about ten hours had elasped since the first *x*-ray examination, another exposure was made, because of the remark-

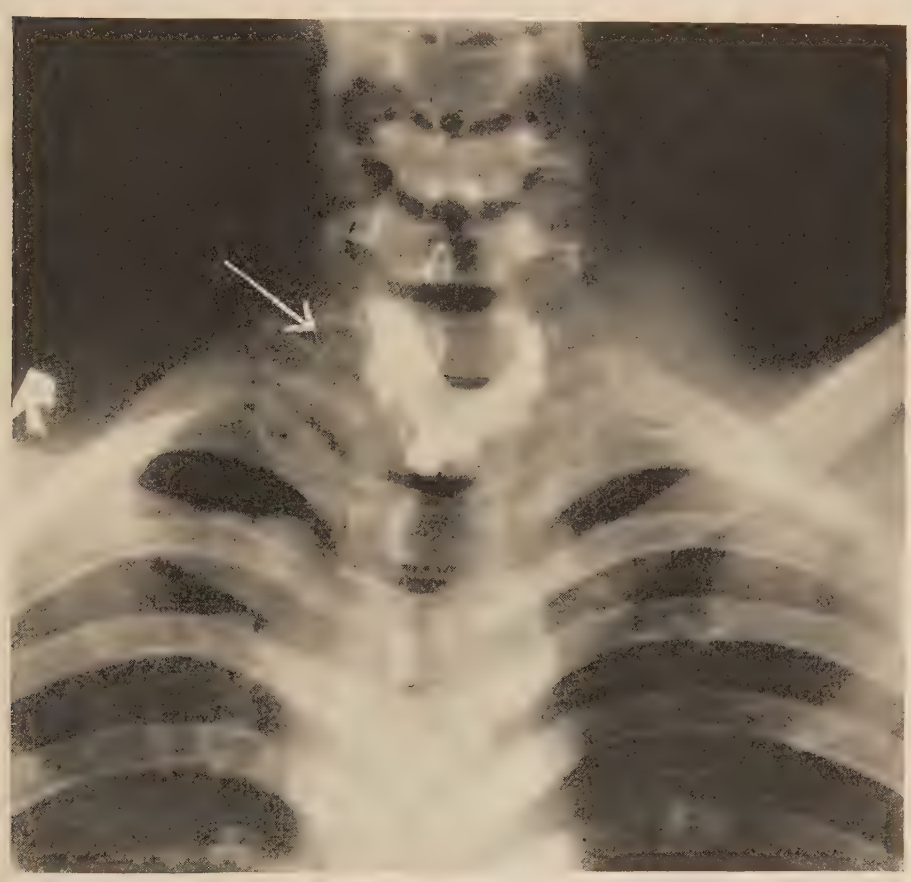

FIG. 1.—Tooth-plate in esophagus. Removed by esophagotomy.

able fact that many irregular foreign bodies, including dental plates, are known to have passed through the entire alimentary canal without injury to the patient. Lediard[1] records a case in

[1] Clin. Soc. Trans., xviii, 297.

which the dental plate appeared at the anus nineteen days from the date of impaction. In our patient the foreign body had not changed its position. Therefore it was decided to proceed with the operation of esophagotomy.

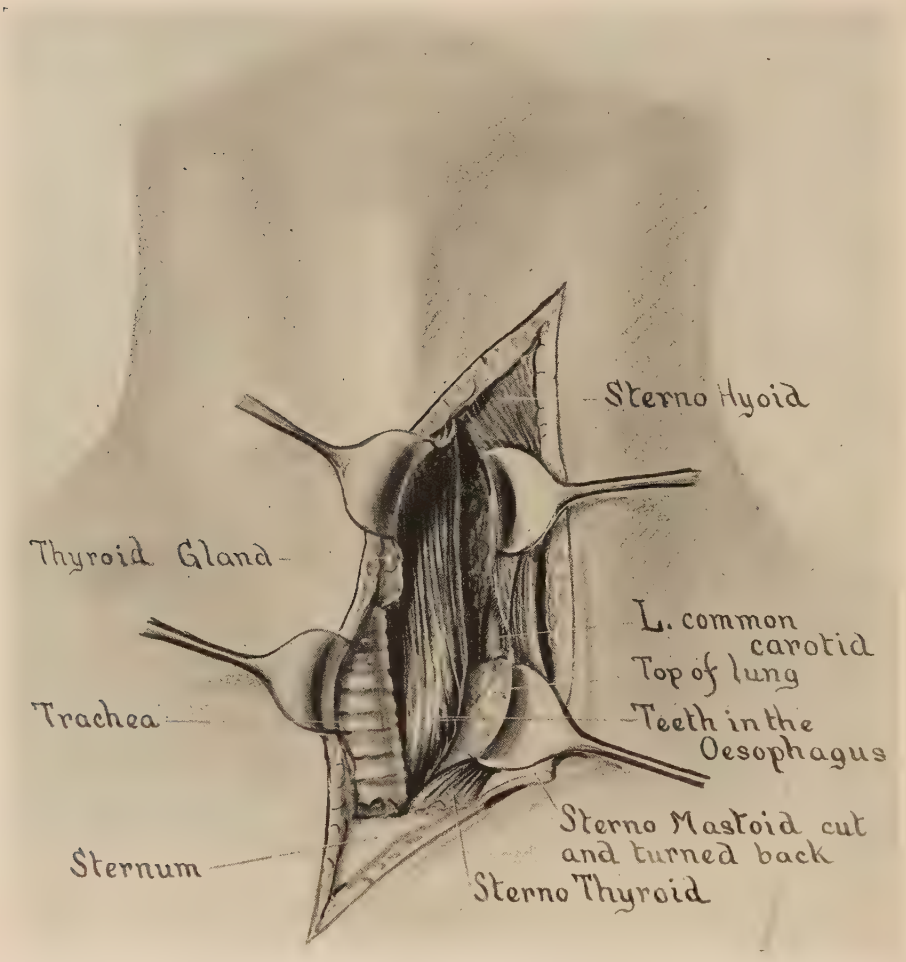

FIG. 2.—Structures met in approaching the esophagus.

The patient was etherized and placed in the Fowler position, with head extended and turned to the right. The skin was

painted with iodin, after which an oblique incision was made following the anterior border of the left sterno-cleido-mastoid muscle and carried down over the sternum for a distance of 5 cm. (Fig. 2). Extending the incision down over the sternum proved advantageous in providing the maximum amount of

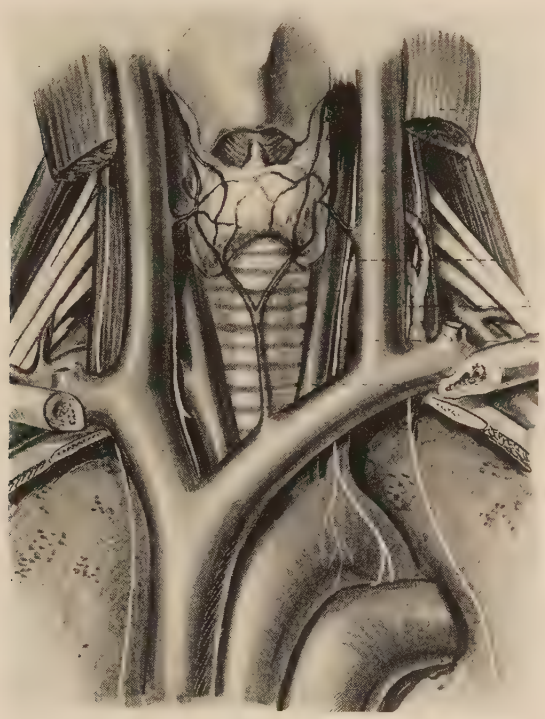

FIG. 3.—Deeper structures met in approaching esophagus.

room where it was needed most. After dividing the cervical fascia, approach to the esophagus was made through the lower carotid triangle, guarding against injury to the structures contained therein. The sternal origin of the sternomastoid was severed and the muscle freely mobilized. The carotid sheath with its enclosed vessels was exposed, as these structures emerged from the superior thoracic aperture (Fig. 3). The sternomastoid

and large vessels were drawn to the left and the trachea to the right, with retractors, the thyroid gland showing plainly over the trachea (Fig. 2). Care being taken to avoid injury of the recurrent nerve (Fig. 4), the loose cellular tissue was separated

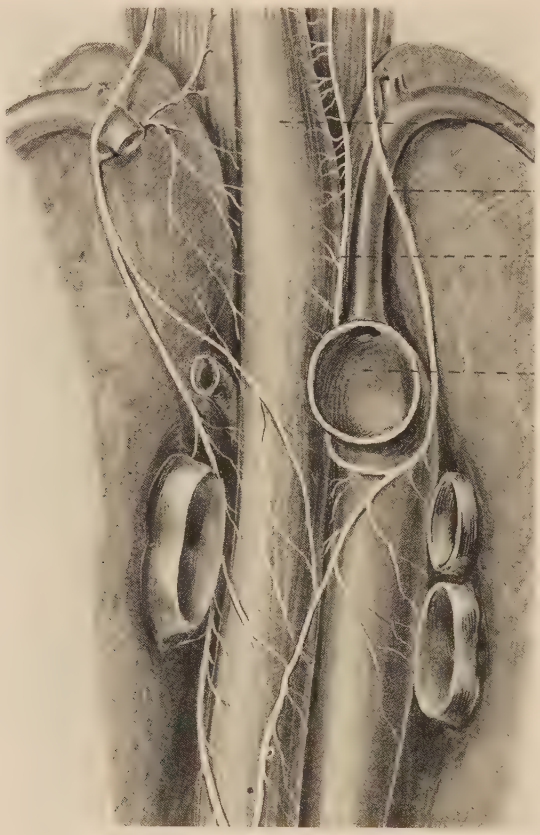

FIG. 4.—Relations of recurrent laryngeal nerve to arch of the aorta and esophagus.

by a method of blunt dissection and the esophagus exposed. At this point the pleura was nicked and disturbed the even course of events. With this repaired the location of the dental plate in the esophagus could be defined readily. Although the foreign

body was fixed there was no evidence of puncture. Before incising the esophagus a small sponge soaked in 70 per cent alcohol was packed in the wound long enough to afford some protection against infection. The vertical incision in the esophageal wall was made somewhat far back in order to avoid the recurrent

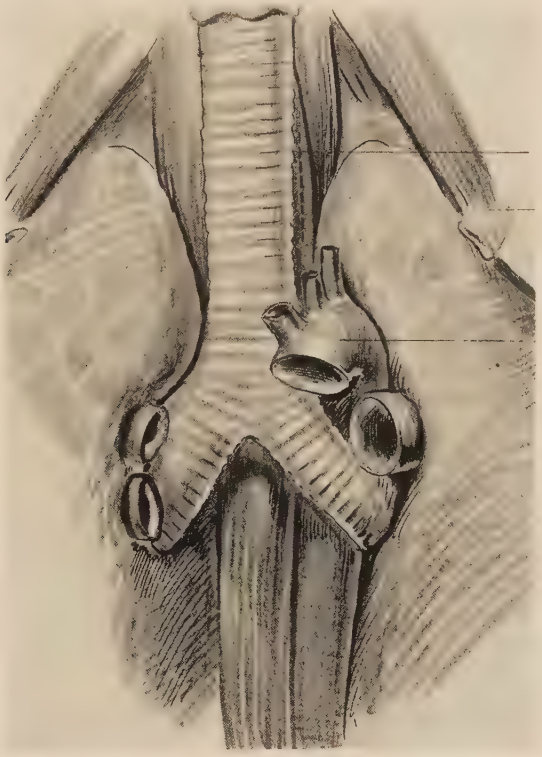

FIG. 5.—Mediastinal space showing proximity of parietal pleura to esophagus.

nerve. Through a 4 cm. opening (Fig. 5) it was with considerable difficulty that the dental plate was dislodged and extracted, so tightly was it embraced by the esophageal fibers. This experience was not unique, for Mr. George Lawson,[1] in a similar operation, found the plate so firmly fixed into the wall of the esophagus

[1] Clin. Soc. Trans., xviii, 292.

that it required division with bone forceps before its removal was possible.

The opening in the esophagus was then closed with a double layer of interrupted sutures of fine silk. A rubber tissue drain was placed in the lower angle of the wound. The skin sutures, used only at the upper angle, were removed on the fourth day. The patient was nourished by means of a Rehfuss tube. A small esophageal leak appeared on the seventh day. This closed in three days. He was discharged on the sixteenth day. There remained only a superficial granulating area. Deglutition was natural and the voice remained normal. At the end of six months he appears to have no stricture of the esophagus and suffers no inconvenience from the scar.

The dangers of esophagotomy for foreign bodies are by no means negligible. The primary risk of the operation is considered to be large. The chief causes of death are hemorrhage, shock and infection. Injury to the recurrent laryngeal nerve is not uncommon. Mr. Cock[1] reported a case of this sort. The patient was a singer. As a result of this accident, his fine tenor voice was replaced by a bass. Stricture of the esophagus may also follow the operation. The mortality, however, cannot be considered very high among the surgeons who first performed this operation. Of 135 esophagotomies reported by Egloff,[2] dating from the first case by Goursald in 1738 to 1894, 100 recovered. Of operations performed in the first three days, 46 showed a mortality of 19.5 per cent. Most of these operations were performed by German surgeons.

It is doubtful if the high mortality, 20 per cent to 42 per cent, referred to by Chevalier Jackson[3] represents the results of this operation in the hands of experienced surgeons under favorable conditions. It is more probable that this high mortality rate can be attributed to "overtreatment" in attempts at extraction by the many so-called "justifiable" procedures with probang and

[1] Jacobson and Steward, i, 563.

[2] Beiträge zur klin. Chir., 1894, p. 143.

[3] Ibid., p. 185.

esophagoscope so commonly advocated as first aid. Even under the guidance of skilled hands, any prolonged instrumentation of the esophagus must react unfavorably upon the chances of success by esophagotomy. Time, which passes rapidly for the operator, is long and wears heavily upon the patient. Only the skilled endoscopist knows when to stop in the use of conservative methods. When the physician or laryngologist is convinced, after a multiplicity of maneuvers, that he cannot extract the foreign body, the patient may be fatally injured. The neck from its external aspects may not present ominous signs, but danger lurks within. Trauma and exhaustion have done their part, and surgery completes an ignoble record. When the foreign body is large, irregular and impacted, will there not be a better chance for the patient's life if esophagotomy is done by a surgeon familiar with the anatomical parts to be met, than if extraction by mouth is undertaken by one who does not possess the proper instruments and has not thoroughly mastered the technical difficulties which the procedure entails?

These accidents happen many hundreds of miles from endoscopic clinics, yet there are few regions where a capable surgeon cannot be reached within fifty miles. Whenever there is a possibility that the open operation may have to be done, would it not be more logical to study the case from every angle with a view to selecting the safest method available? That method may not represent the last word in the technical features pertaining to the treatment of these cases, but on certain occasions the first-born method may be found more expedient, fully as trustworthy and even less precarious. If esophagotomy is undertaken only after "bloodless" methods fail, necessarily the mortality must be very high. Under favorable conditions it should not exceed 5 per cent.

In order that the open operation may have the best chance for success when it is indicated, it is essential that no preliminary probing of any sort by mouth be allowed. That feature more than any other contributed to the success of the case which I have reported. Surgery in this as in the treatment of many other

diseases must not be chosen as a last resort if the aim is to save life. Although well-nigh obsolete, I believe esophagotomy is still a precious procedure in wisely chosen cases of foreign bodies in the esophagus, and the success of this operation is never enhanced by trial measures, which shatter a patient's stamina and induce prostration. To have its fair chance in this particular dilemma, esophagotomy must be given an untrodden field at the start.

DISCUSSION

Dr. Willy Meyer, New York:

Dr. Truesdale certainly acted wisely in considering his case from all angles and putting the life of the patient in the first line. Whether in such a case a surgeon should step in or a specialist will largely depend on whether the latter is available. Certainly, if surgery is necessary no preliminary treatment should be done, because, after all, the eye must be our chief aid, that is, the eye through the esophagoscope or the surgeon's eye after the esophagus has been opened.

Not long ago we listened, with great interest, to a chalk talk by Dr. Chevalier Jackson, at the joint meeting of the Philadelphia Academy of Surgery and the New York Surgical Society. I would like to repeat here what I said then: "There can be no question to my mind that in a short time there will be specialists in the so-called peroral endoscopy everywhere. Today, almost every hospital of standing has its own endoscopist, or a number of hospitals have joined hands and have given one man this work. I am sure the time will soon be at hand when the physicians of a number of rural communities will join hands in the employment of a well-trained specialist in this work." Needless to say, however, esophagotomy will retain its place in the surgical field; the careful weighing of the pros and cons shows the good surgeon and the good specialist.

With reference to the danger connected with the examination, there exists a distinct difference regarding bronchoscopy and esophagoscopy. I have never seen a fatality after bronchoscopy, and I am sure that other hospitals had the same experience. It is different with esophagoscopy; there we have seen deaths. I believe it is often due to the method employed. Without meaning to criticize our specialists, I would say that they, of course, introduce the bronchoscope as far as they conveniently can, and then they look in. It is wrong to do the same with the esophagoscope. What did we do with the examination of the urethra many years ago? We introduced the urethroscope just through the external meatus, and then, looking at the inside of the canal continuously, gradually and slowly pushed it forward. Thus a small tumor could be seen, a stricture clearly recognized, etc. The esophagoscope should be similarly handled.

Dr. Howard Lilienthal, New York:

I believe that patients with foreign bodies in the esophagus ought to have the benefit of an *x*-ray examination and then that the esopha-

goscope should be employed by a man who is experienced in its use. The esophagoscope is a much more dangerous instrument than the bronchoscope. Ordinarily we think of esophagoscopy as a much simpler procedure than bronchoscopy. This is not so. The reason is not because of the instrument, but because of the anatomy of the esophagus, which is a pretty "mushy" sort of an organ compared with the bronchus.

I agree with what Dr. Meyer has said about posterior mediastinotomy if the services of an endoscopist are not available, but, of course, all foreign bodies do not go as far as the mediastinum, as did this one of Dr. Truesdale's. In these cases there is the necessity for quick and skilful action on the part of the surgeon. One can do harm by injudiciously trying to work from above, especially if the impacted body is a denture with perhaps sharp points, because it may easily perforate the esophagus and produce mediastinitis. It is very important that we should use every means to orient ourselves in locating the esophagus. I have devised an instrument that can be very quickly and easily improvised, and because of its simplicity I want to show it to you. All you need is a pocket battery with lamp, or a cystoscopic lamp for that matter. The lamp is fastened into the end of a stomach tube, the wires passing through the tube to the battery held in the hand. By this transmitted light the esophagus is easily found and the veins and nerves running over it are plainly seen and may be avoided.

THE EFFECT OF PRESSURE ON ARTICULAR SURFACES IN PYOGENIC AND TUBERCULOUS ARTHRITIDES AND ITS BEARING ON TREATMENT

By D. B. PHEMISTER, M.D.
CHICAGO, ILL.

It was recognized that pressure played a role in the destruction of articular surfaces in arthritis long before the bacterial nature of infection was known. But since the time that pyogenic and tuberculous arthritides were differentiated bacteriologically, the differences in the effects of contact and pressure on the articular surfaces in the two conditions have not been fully elaborated. In fact, it is too generally considered that they are much the same in the two conditions, that the effects are destructive, and that in both the articular surfaces are destroyed first and most extensively at the points of contact and pressure of opposing articular surfaces. Examination of a series of specimens showing each disease in its various stages demonstrates that this is not the case. The changes as influenced by contact and pressure are more accurately described for pyogenic than for tuberculous arthritis. Koenig's[1] work on tuberculous arthritis is the most extensive and exact, but certain inaccuracies may be found in it pertaining to the persistence of cartilage longer at certain points than at others and to bony invasion and necrosis. Also, knowledge as to proteolytic activities in pyogenic and tuberculous infections has not been utilized in explanation of the changes seen in articular cartilages in pyogenic and tuberculous joints. In studying the effects of contact and pressure a distinction should be made between those produced on the articular cartilage and those produced on the underlying bone.

In acute pyogenic arthritis it is found that when there is an effect from contact and pressure, it is to help to destroy articular cartilage in the regions of contact of opposing articular surfaces. On the other hand, in tuberculous arthritis the effect, except in the later stages, is protective and articular cartilage is usually preserved longest at the points of contact and pressure, while the first evidences of destruction are found over the free surfaces. The differences are present because the agents which attack and destroy articular cartilage are very different in the two processes. These observations have been made mainly on adults and on the knee-joint, where, because of large areas of both free and opposed articular surfaces, conditions are favorable for contrasting the effects of pressure and lack of pressure. They hold, in varying degrees, for other joints and for children. In both pyogenic and tuberculous infections the articular cartilage is involved secondarily, the primary infection being either in the synovia or in the bone.

Pyogenic Arthritis. Effects of Pressure on Cartilage. In pyogenic arthritis the articular cartilage may not become involved if the infection is mild, but if the infection is severe, cartilage will be killed, and it is generally killed first and most extensively at the points of contact and pressure of opposing articular surfaces. This is undoubtedly related to the unfavorable conditions for nutrition produced by pressure. The amount of necrosis on surfaces that are not pressed upon varies. In some cases it is slight, while in others the entire cartilage may be killed. Whatever happens on one side of the joint usually happens on the other. If cartilage is killed in the opposed regions on one side, it is usually killed to the same extent on the other side. Dead cartilage in pyogenic arthritis is usually destroyed in a comparatively short time. It is broken down partly by the absorptive action of granulation tissue, partly by erosion of opposing articular surfaces, and partly by the digestive action of proteolytic ferments in the exudate of the joint. These ferments are derived very largely from the polymorphonuclear leukocytes, to a slight extent from the bacteria. Where cartilage

is killed in its entire thickness, granulations form beneath its attached surface and rapidly detach it by absorption of both cartilage and bony cortex at their junction. Where only a superficial layer of cartilage has been killed in unopposed regions, it is removed mainly by digestion, but granulations may also grow from the margins over the unopposed surfaces and absorb the dead layer.

A series of experiments has been performed to test the rapidity with which articular cartilage is digested by a pyogenic exudate *in vitro*. When pieces of articular cartilage are immersed in pus

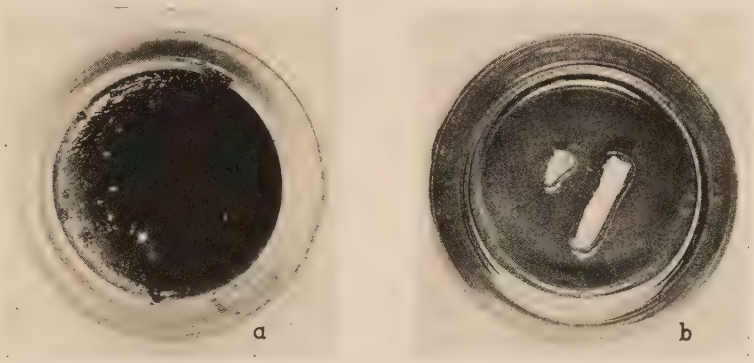

FIG. 1.—Equal amounts of articular cartilage in staphylococcus pus (a) and in tuberculous pus (b) after incubation at 55° C. for ten hours; cartilage digested in (a) and undigested in (b).

produced by any of the pyogenic microörganisms and the mixture incubated at a temperature of 55° C., so that proteolytic action is augmented, the cartilage is digested in from three to twenty-four hours, depending on the concentration of the pus. Fig. 1 shows two watch-glasses, each of which at the beginning of the experiment contained two pieces of fresh articular cartilage and underlying bony cortex of the size shown in (*b*). Staphylococcus pus was added to (*a*) and pus from a tuberculous cold abscess to (*b*). After incubation for ten hours the articular cartilage in (*a*) was completely digested and particles of bone sand were the only solid materials remaining. The pus was more

liquid than at the beginning of the experiment, because of the breaking down of its proteids and of the leukocytes themselves. At a temperature of 55° C. bacterial action is suspended, so that the digestion was produced by existing ferments in the pus. That the ferments are derived very largely or wholly from the polymorphonuclear leukocytes in the exudate is shown by the fact that relatively little or sometimes no cartilage is digested when it is incubated in a suspension of pyogenic bacteria. Experiments have demonstrated that a variable amount of proteolytic ferment is formed by different organisms, as shown by their ability to break down tissues and to liquefy such media as gelatine and blood serum. According to Bittrolff,[2] it is small in amount with most pathogenic forms. Heavy suspensions in normal salt solution of some strains of Staphylococcus aureus were found not to digest articular cartilage, while others digested it to a slight degree. In the experiment shown in Fig. 2, equal sized pieces of articular cartilage were placed in the tubes. Tube (1) contained normal salt solution, tube (2) a suspension of staphylococci in a concentration of 15,000,000 per cubic millimeter, and tube (3) a suspension of 40,000,000 per cubic millimeter. They were incubated at 55° C. for six days, and there was no reduction in size of the pieces of cartilage. Microscopic examination of sections showed cartilage well preserved in the piece incubated in salt solution and only very slight breaking down of cells and vaculation of intercellular substance in the pieces incubated in the staphylococcus suspensions. The experiments of Bittrolff,[2] Cacace,[3] and others have shown that proteins may be split by the action of bacterial ferments into albumoses, peptones and amino-acids.

Effects of Pressure on the Bone. In pyogenic arthritis the changes in the bone bordering on articular surfaces vary according to the point of primary infection. In primary arthritis with secondary involvement of cartilage, the articular cortex of bone is nearly always destroyed in those regions where the entire thickness of articular cartilage is broken down. Consequently, it is destroyed oftenest and most extensively at the points of

pressure. An inflammatory reaction is seen in the adjacent layer of spongy bone of these regions, but deep invasion producing osteomyelitis and sequestration, even at the points of pressure, is rare. In infected penetrating wounds of joints with associated joint fracture and osteomyelitis, death of detached bony frag-

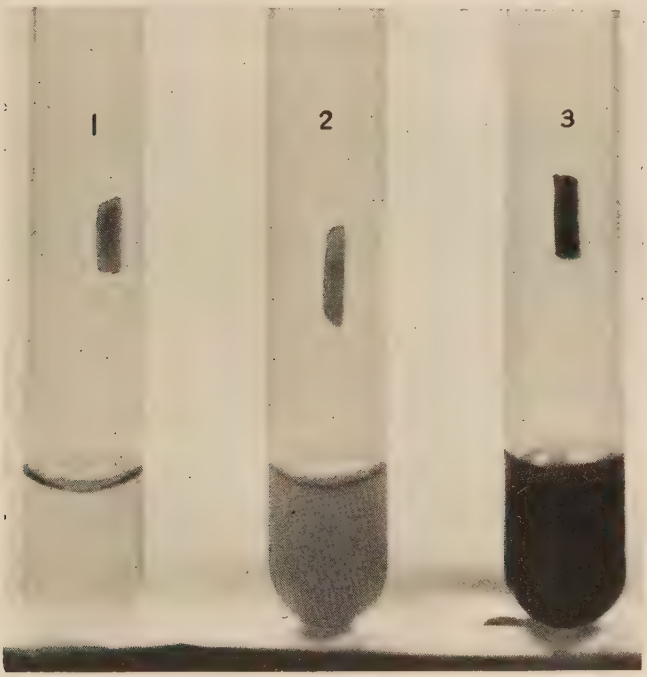

FIG. 2.—Equal sized pieces of articular cartilage incubated at 55° C. for six days in normal salt solution (1) and staphylococcus suspensions (2) and (3). No change in (1) and only slight microscopic changes in (2) and (3), showing little digestion by the bacteria.

ments is common. When the primary infection is an osteomyelitis which spreads into the epiphysis and the joint, necrosis and sequestration of bone bordering on the articular surface is not uncommon, and even here, as in the head of the femur, it may be greatest in the weight-bearing region. When bone and overlying articular cartilage are both killed, the cartilage rapidly

disappears by the digestive action of ferments; but the dead bone with its layer of articular cortex may persist and be separated as a sequestrum. Fig. 3 shows a photograph of the articular surfaces of the bones of the knee and ankle, which were involved in arthritis by direct extension from staphylococcus osteomyelitis of the entire shaft of the tibia in a twelve-year-old boy. The infection in the knee was purulent and was drained on the

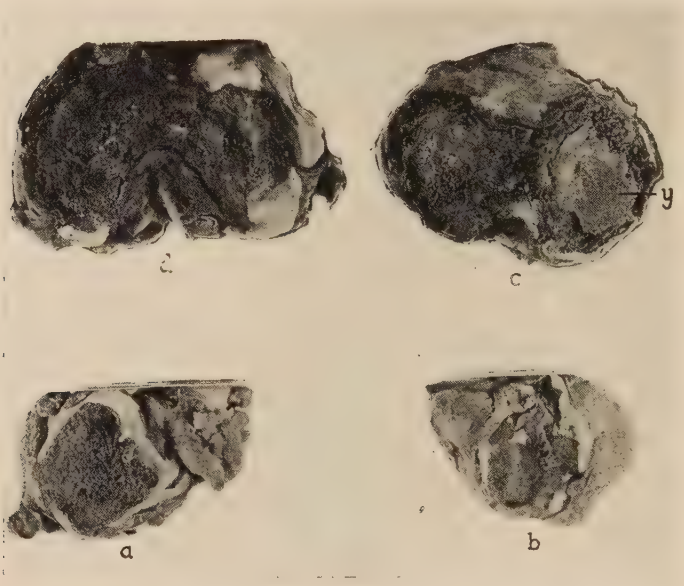

FIG. 3.—Joint surfaces in pyogenic infections of knee and ankle. Cartilage destroyed at pressure points and largely preserved where not compressed on astragalus (a), lower end of tibia (b) and femur (d). Cartilage all destroyed on upper end of tibia (c). Cortical sequestrum (y).

fifty-fourth day, when the patient was admitted to the Presbyterian Hospital. That in the ankle was less severe and had subsided without drainage when the limb was amputated above the knee on the seventy-fifth day of the illness. Articular cartilage and cortex of bone were almost completely destroyed at the points of contact and pressure of the astragalus (*a*) with the tibia (*b*), but were preserved about the sides of the joint on the

astragalus, tibia and fibula, where there was little or no pressure. At the knee, where the arthritis was severe, the destruction of articular surfaces was more marked. The infection had involved a part of the upper epiphysis of the tibia and had killed articular cartilage and underlying cortex of both tibial tuberosities (*c*). The cartilage had been destroyed by digestion, but portions of articular cortex were present as disk-like sequestra. The one on the internal tuberosity is shown *in situ* at (*y*) in Fig. 3. The femur (*d*) showed complete disappearance of articular cartilage and of underlying cortex on the condyles at the points of contact and pressure with the tibia and partial destruction of cartilage and cortex at the point of contact with the patella, which latter structure had also lost its cartilage, excepting remnants about the periphery. The cartilage was largely preserved on the remaining free surfaces, anteriorly between the points of contact with the patella and tuberosities and posteriorly on the condyles, but its surface was mostly uneven from superficial destruction. This case illustrates well the disappearance of cartilage and cortex where pressed upon by opposing articular surface, the survival of cartilage in the regions of the joint that are free from pressure, and the preservation of bony cortex and disappearance of cartilaginous covering on an articular sequestrum.

A comparatively mild acute arthritis may involve articular cartilage at the points of greatest pressure, which will lead to bony ankylosis unless measures are introduced to combat it. Fig. 4 shows a photograph of the articular surfaces of the right knee five months after the onset of a hematogenous arthritis secondary to erysipelas of the left thigh. There was an effusion at the onset and aspiration yielded a turbid serous fluid, from which a hemolytic streptococcus was grown. The infection subsided spontaneously after six or seven weeks, leaving the joint with only a small range of motion. Extension was not applied to the limb, nor was the joint mobilized. The limb was amputated because of recurrence of an old staphylococcus osteomyelitis of the upper end of the femur with coxitis. Dissection showed destruction of articular cartilage and cortex at the points

of contact of external condyle (*a*) and external tuberosity (*b*), which is the region of greatest pressure in the knee-joint, and a bony bridge was in process of formation here. The articular cartilage was preserved elsewhere on the tibia and femur and

FIG. 4.—Ankylosis five months after mild undrained arthritis of knee. Cartilage destroyed only at points of pressure of external condyle (a) on external tuberosity (b). Fibrous ankylosis elsewhere.

on the patella, but there was extensive fibrous ankylosis present in these regions. The extreme degree of destruction of cartilage

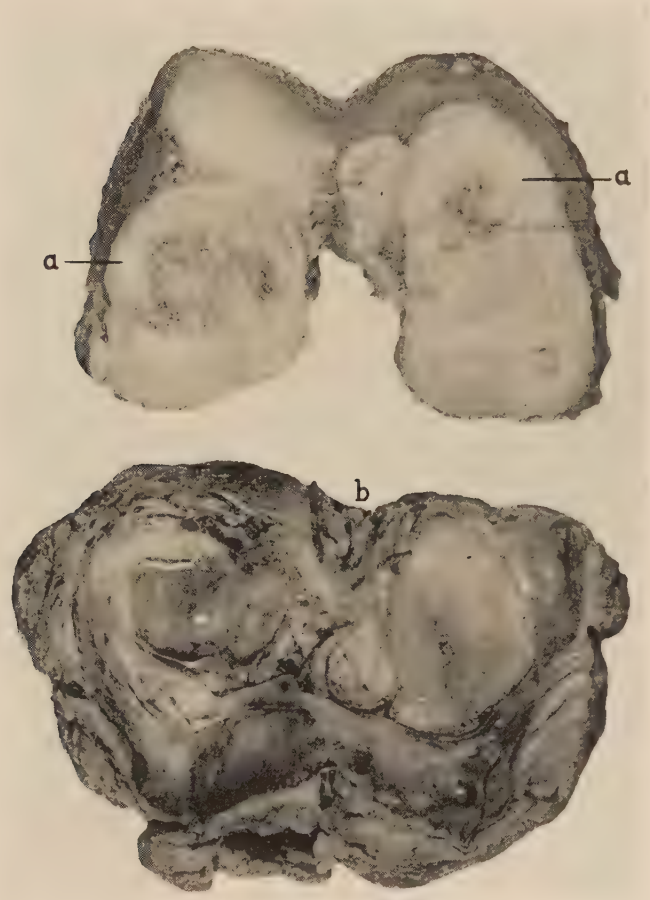

FIG. 5.—Healed undrained pyogenic arthritis of knee. Roughened healed areas where cartilage is destroyed at points of pressure on condyles of femur (a); cartilage on tibia (b) little changed.

and the fibrous ankylosis resulting from the comparatively mild arthritis were no doubt related to the fact that the knee was

neither mobilized nor extended during any part of the course of the infection.

In moderately severe pyogenic arthritis it may rarely happen that articular cartilage and underlying cortex are destroyed at the points of pressure on one side of the joint and persist on the other. The presence of cartilage on one side lessens the liability to the development of ankylosis, and there may be healing with

FIG. 6.—Section through external condyle shown in Fig. 5, showing layer of articular cartilage (a) destroyed at point of greatest pressure and overgrown by partly ossified reparative tissue (b) growing out from the bone.

preservation of considerable motion. The area of destroyed articular surface is repaired by the outgrowth of granulation tissue from the underlying bone, and it may overgrow the surrounding cartilage, producing a ridge about the margins of the defect. These granulations change into fibrocartilage and may ossify in their deeper portions, producing osteophytes on the articular surface. Fig. 5 shows the articular surfaces of a man's

knee, resected one year after spontaneous healing of a mild sero-purulent arthritis, resulting from extension of a staphylococcus

FIG. 7.—Roentgenogram of knee shown in Fig. 5. The ossified reparative tissue overgrowing the articular cartilage about margins of the regions of destruction casts disc-like shadows in cartilage spaces between condyles and tuberosities.

osteomyelitis of the tibia. The articular surfaces had been destroyed at the points of contact on the condyles of the femur,

but not on the tibia, where they were somewhat protected by the marginal support of the semilunar cartilages. The damaged areas on the condyles had been repaired by a layer of fibrocartilage, the margins of which were raised and in places overhanging. A sagittal section through the lateral condyle showed that articular cartilage and cortex had been destroyed at the point of greatest pressure and that reparative tissue had grown out from the underlying bone, filling the defect and overlying the margins of the surrounding cartilage (Fig. 6). The superficial portion of this tissue was fibrocartilage, while the deeper portion has ossified. The shadow cast in the roentgenogram by the new bone overlying the remaining articular cartilage gives the appearance of a bony disk interposed in the cartilage space of the joint between condyle and tuberosity (Fig. 7). If cartilage and cortex break down on both sides of the joint, it is extremely difficult to avoid the occurrence of bony ankylosis.

Tuberculous Arthritis. Effects of Pressure on Cartilage. In tuberculous arthritis the point of primary infection is either in the bone or in the synovial lining, and the articular cartilage becomes involved secondarily. The inflammatory reaction is not sufficiently severe to kill articular cartilage *en masse* in the early stage of the disease, as is so often the case in pyogenic arthritis. Tuberculous synovitis usually runs for months and is well established before there is any sign of destruction of articular surfaces. The articular cartilage is then first killed and absorbed by the direct attack of tuberculous granulations, which grow onto it from the surrounding synovia. They attack it first along its free surfaces and about its margins, where they can readily get at it. The cartilage is protected from the attack of tuberculous granulations in the regions of contact and pressure of opposing surfaces in the joint, and the destruction may be very marked along the free surfaces and about the margins before there is any change in the cartilage of the contacted regions. This is illustrated by Fig. 8, which shows the articular surfaces of the resected femur and tibia in tuberculosis of the knee in an eighteen-year-old boy. There was marked tubercu-

lous synovitis, and granulations had attacked the cartilage of the tibia about its margins; but the central portions of cartilage on either tuberosity were free from attack, as were the surfaces of the condyles of the femur with which they come in contact. The surfaces of contact of the patella and femur were also free. Granulations covered the free cartilaginous surface of the femur

FIG. 8.—Tuberculosis of knee. Cartilage preserved where patella and tuberosities come in contact with femur and partly destroyed along unopposed surfaces of tibia and femur.

between its points of contact with the patella and tibia and on the posterior surface of the condyles, and had absorbed most of the thickness of the cartilage. The layer of granulations is seen intact between the patellar surface and internal condyle, but it has been removed, leaving a grooved and roughened surface, between patellar and external condylar surfaces.

Destruction of cartilage in the regions of contact and pressure is usually brought about first by subchondral granulations, which may be tuberculous in nature about the periphery of the cartilage, but which are non-tuberculous beneath the more centrally located portions of cartilage. There they consist of capillaries, fibroblasts

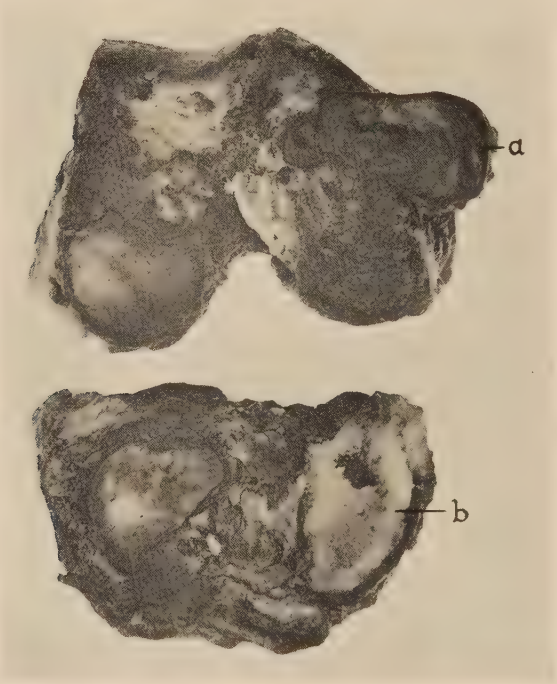

FIG. 9.—Tuberculous knee of two- and one-half-years' duration. Cartilage largely preserved at points of contact of patella and tibia with femur and destroyed elsewhere in joint. Contacting cartilage undermined on mesial condyle (a) and mesial tuberosity (b).

and round cells, and in the capillary loops that invade and absorb the cartilage polymorphonuclear leukocytes may be seen. They gradually detach the cartilage by absorbing articular bony cortex and deeper portions of cartilage. As in pyogenic arthritis, whatever happens on one side of the joint usually happens on

the other. If cartilage is preserved or eroded or undermined on one side, the same condition obtains at points directly opposed. These changes are well illustrated in Fig. 9. It shows the articular surfaces of the femur and tibia of a man whose knee was resected because of tuberculous arthritis of two and a half years' standing. Cartilage is attached and preserved on the central portions

FIG. 10.—Lateral view of joint in Fig. 9 with sagittal section through lateral condyle, showing cartilage preserved in regions where femur came in contact with patella (a) and tibia (b). Cartilage destroyed elsewhere.

of the surfaces of contact of femur with the patella and of lateral condyle with lateral tuberosity, which is the point of greatest pressure in the joint. This is the region in the knee-joint where cartilage usually persists longest. It is detached and thinned by undermining granulations on the mesial condyle and tuberosity. It has been destroyed everywhere else in the joint except in these regions of contact, and they show marginal absorption. Fig. 10 is a side view of the same specimens, with a sagittal sec-

tion through the lateral condyle. It shows the preservation of cartilage and of articular cortex of bone at the points of contact of patella and of tibia with femur and the loss of cartilage and of articular cortex in the unopposed regions. The preservation

FIG. 11.—Side view of roentgenogram of joint in Fig. 9, showing cartilage space and articular cortex preserved in regions of contact of external condyle and tuberosity and of patella and femur, and articular cortex absent elsewhere.

of cartilage space and of articular cortex in the opposed regions is demonstrable in the roentgenogram taken before operation and shown in Fig. 11. The first roentgenographic evidences of destruction of articular cortex in tuberculosis of the knee are usually seen along the unopposed surfaces and about the margins

of the articular surfaces. Preservation of both the normal width of cartilage space and shadow of cortex in the regions of contact and pressure is in favor of tuberculous arthritis, while loss in those regions with preservation elsewhere in the joint is in favor of pyogenic arthritis.

In these joints whose articular surfaces have like contour and in which cartilage fits snugly against cartilage throughout, leaving little or no unopposed surface, the loosening and destruction of articular cartilage is carried on mainly by the action of undermining granulations. This is true of the hip- and ankle-joints, where, on opening the joint at the right stage, the cartilages may be found completely detached and considerably thinned from beneath, while there is little or no evidence of destruction along their free surfaces. After the disease is well advanced and destruction is extensive in the unopposed regions of the joint, the remaining cartilage in the opposed regions may become necrotic. Cartilage is also killed *en masse* when the undermining granulations destroy its bony connections. In fact, the explanation of the development of the non-tuberculous subchondral granulations may be the necrosis of the cartilage without the entrance of tubercle bacilli into the subchondral regions. The granulations then form as a foreign-body reaction to absorb and sequestrate the dead cartilage.

The experimental work of Nussbaum[4] supports the theory that articular cartilage, except the deepest layer, receives its nutrition from the synovial fluid. If the joint fluid continues to be the source of nutrition for the cartilage in tuberculous arthritis, it is understandable how, after the disease is well advanced, the altered exudate no longer furnishes adequate foodstuffs for cartilage, and it dies *en masse* from lack of nourishment, as well as from the effect of tubercle toxins.

Dead cartilage, whether attached or detached, may persist in the joint for incredibly long periods, because of the absence of active proteolytic ferments in the tuberculous exudate. Pressure and motion erode and destroy dead cartilage. But when these are absent or slight, detached disks may persist for many months

and dead cartilage may stand for years on articular sequestra, since granulations cannot get at its base to absorb it and there are no proteolytic ferments in the exudate to digest it. Edward Mueller,[5] Jochmann,[6] Opie and Barker,[7] and others have shown that exudates in tuberculous processes, including cold abscesses, contain practically no active proteolytic ferments, and we know that tuberculous granulation tissue is killed by coagulation necrosis, coagulins being formed by the tubercle bacilli (Ruppel,[8] Schmoll[9]), which precipitate the soluble colloids of the cells. The caseous areas persist for extremely long periods, because of the absence of active digestive ferments.

No published studies were found on the effect of tuberculous exudates on dead articular cartilage in relation to the persistence of cartilage in tuberculous joints. The digestive action of tuberculous exudates on cartilage was tested *in vitro*. Cold abscesses were aspirated and cartilage was incubated at a temperature of 55° C. in the fluid obtained. The tuberculous fluid coagulated in a short time, due to its richness in albuminous substances and both articular cartilage and coagulum persisted, after several days of incubation, without any signs of digestion. This shows the absence of active proteolytic ferment in the exudate. The tubercle bacillus is found to contain no proteolytic ferments and its toxins destroy the autolytic enzymes of the dead tissues. Consequently, there is no autolysis of the dead cartilage. The leukocyte present in the tuberculous granulations and exudate is mainly the large mononuclear cell. It contains some proteolytic ferment, which is active in acid media, but not in tuberculous exudates, which are always alkaline in reaction (Opie and Barker[7]).

Fig. 1 (*b*) shows cartilage unchanged and tuberculous fluid coagulated after ten hours of incubation. Fig. 12 shows test-tubes 1, 2, 3 and 4, in which were placed equal-sized pieces of cartilage. To them were added, respectively, salt solution, tuberculous pus, staphylococci suspended in salt solution, and staphylococcus pus. They were incubated for forty hours at 55° C. The cartilage remained unchanged in the salt solution and in the tuberculous

exudate, and the latter coagulated; cartilage in the bacterial suspension was slightly reduced in size, while that in the staphylococcus pus was completely digested. The pus in tube 4 was much more liquid than at the beginning of the experiment.

FIG. 12.—Cartilage digestion experiment. Equal sized pieces of cartilage incubated at 55° C. for forty hours in normal salt solution (1), tuberculous pus (2), staphylococcus suspension (3) and staphylococcus pus (4). Cartilage unchanged in (1) and (2), slightly changed in (3) and completely destroyed in (4). Tuberculous pus (2) was coagulated and staphylococcus pus (4) was liquefied.

THE EFFECT OF PRESSURE ON THE BONE IN TUBERCULOUS ARTHRITIS. Secondary invasion of the bone in tuberculous arthritis does not usually occur as long as the articular cartilage remains little disturbed. The non-tuberculous granulations which undermine the cartilage as the disease advances are super-

ficial and rarely invade the bone to any appreciable extent. The tuberculous granulations which undermine the margins of the cartilage and attack the bone in regions where cartilage has been completely destroyed may invade the bone to some extent, absorbing it, with resultant pits and grooves in the bony surfaces. Secondary invasion of bone resulting in necrosis and sequestra is of rare occurrence in the regions of the joint that are not subjected to pressure. While cartilage is usually protected and preserved longest at the points of pressure, the bone in these regions, after joint cartilage is largely or entirely destroyed, does not fare likewise. On the contrary, it is more subject to involvement than that in any other part of the joint. When cartilage has disappeared, the pressure and friction of bony surfaces may produce extensive bony erosion, as of femoral head and acetabulum at the hip, with pathological dislocation. Occasionally in weight-bearing joints, particularly the knee, there may be sclerosis of the bone to some depth and polishing of the bare, bony articular surfaces at the points of weight-bearing. At an advanced stage there is not infrequently extensive secondary invasion of bone in the zones of pressure either before or after their coverings of cartilage have been completely destroyed. Undoubtedly the damaging influence of pressure is instrumental in producing this invasion, and if the bone is invaded on one side of the joint, it is apt to be invaded on the other side. This results in necrosis of bone, and the areas are so large that the dead bone is usually not absorbed, but is gradually sequestrated. When the condition is bilateral, it produces "kissing sequestra." If cartilage is still present on the articular surface at the time of invasion, it will die and may subsequently be removed by erosion, or it may stand for a very long time, as granulations cannot readily get at it and the exudate does not break it down by proteolysis. The articular cortex is always preserved on the sequestrum, if cartilage is still present at the time of bony invasion. The sequestra usually show some evidence of bone atrophy, which had occurred from disuse before invasion and death of the bone. After death their density remains stationary, while that of the

surrounding living bone is gradually reduced as the result of continued atrophy and absorption. In the roentgenogram such sequestra are recognizable by their density, which is greater than that of the surrounding living bone, and by the presence of a shadow of articular cortex and sometimes of a zone of demarcation. If articular cartilage and cortex have been destroyed before bony invasion, there may be seen in the roentgenogram no sharp outline of the articular surface of the sequestrum. If bony sclerosis and polishing of the surface develop before bony invasion and death, the shadow of cortex and of underlying bone in the sequestrum may be very heavy. In fact, sclerosis may very rarely result in density which is greater than that of normal bone of the region. Occasionally there may be calcification of the necrotic tissue of the cancellous spaces of the dead bone, which may cause the sequestrum to cast a heavier shadow than normal bone of the region.

Fig. 13 is of a roentgenogram taken three years after the onset of tuberculous coxitis in a man, which had produced marked symptoms and had been treated by immobilization during the previous six months. At the top of the joint, which is its point of greatest pressure, there is an area in the head and one opposed to it in the ilium which cast heavier shadows than the surrounding living bone and are separated from it by zones of demarcation. A definite, sharp shadow is cast by articular cortex on each dense area, while the shadows of articular cortex in the rest of the joint are absent. At operation by Dr. Ryerson extensive tuberculous coxitis was found. In the regions casting denser shadows there were two kissing sequestra. On each the articular cortex was preserved, and there was a thin layer of cartilage about the periphery of the much more extensive articular surface of the sequestrum from the head (Fig. 14). The findings indicate that articular cartilage was present on both areas when they were invaded and killed, and that the dead cartilage in the regions of contact of the two surfaces was destroyed by erosion, while that on the unopposed margins of the larger surface of the head was only partly destroyed. On microscopic examination

evidence of slight atrophy of disuse was found in both sequestra, which indicates that an interval of time separated the bony invasion from the onset of the disease. The presence of atrophy within the sequestra, their equal density and their situation at the point of greatest density in the joint are conclusive evidence that they arose from simultaneous secondary invasion.

FIG. 13.—Tuberculosis of hip, showing two kissing sequestra at point of greatest pressure in joint.

The presence of opposing sequestra in the knee-joint has been mentioned by Koenig[1] and Krause,[10] but an incorrect explanation of their development was given. Koenig assumed that one sequestrum represented the area of primary infection and that

the other arose by secondary invasion across the joint after tuberculous arthritis had been established. No mention is made of the fact that they develop only in the regions of greatest pressure in the joint, nor was any comparison made of the patho-

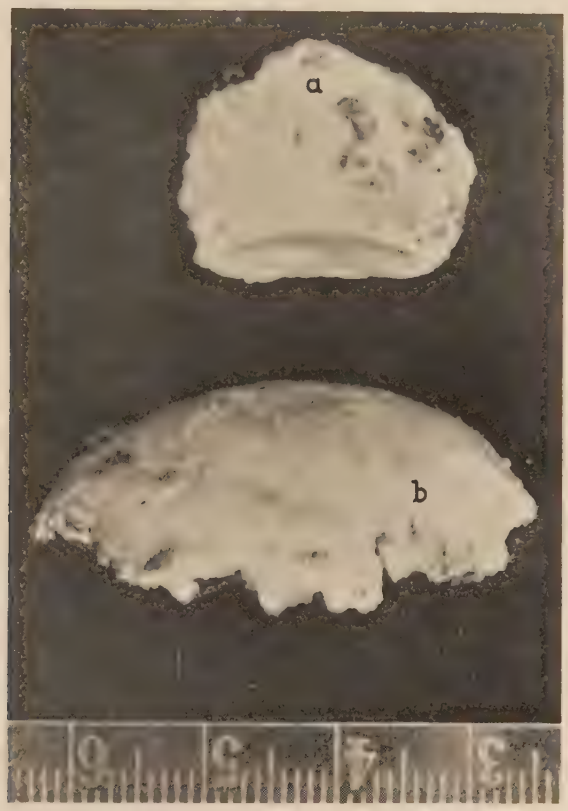

FIG. 14.—Photograph of kissing sequestra shown in Fig. 13, (a) from ilium and (b) from head.

logical changes presented by the two sequestra. In a study of a comparatively limited number of joints operated on for advanced tuberculous disease, I have met with eight instances of kissing sequestra or opposed areas of necrosis in which sequestration was not yet complete. Seven were in the knee, and one, the case

above reported, in the hip. Detailed roentgenological and pathological examinations show that in every instance the bony invasion occurred simultaneously and secondarily on the two sides of the joint. That the lesions developed simultaneously is shown by the fact that the pathological changes are exactly the same on the two sides. They each present the same degrees of density and of destruction. If articular cortex is present on one, it is present on the other also. If dead articular cartilage is found on one, it is found on the other, unless the articular surfaces are of unequal size, in which case cartilage may be absent from erosion where the surfaces of the sequestra come together, but present on the unopposed portion of the larger surface.

That both sequestra arose secondarily is shown by a number of facts. Since the pathological evidence indicates that the two lesions arose simultaneously, it is evident that they are either both primary or both secondary osseous involvements. It is inconceivable that bacteria from the blood stream would so often lodge at the same time in the bone underlying directly opposed surfaces of the joint, and always in the regions of greatest pressure. Microscopic examination of the sequestra showed evidences of atrophy of the bone before its death. In some cases the atrophy was slight, while in others it was very marked and equal to that in the surrounding living bone. The necrotic bony areas may be found attached to the surrounding living bone, with few signs of absorption and sequestration about their margins. This is evidence of recent invasion, and when the dead bone is also atrophic, the secondary nature of the lesions is definitely established.

The shape of an area of necrosis is variable. It may be that of an oval or often that of a low cone with its base bordering on the joint. Since Koenig's publications it has been the custom to regard all cone-shaped areas of necrotic bone with their bases on the articular surface as the result of embolism, clumps of tubercle bacilli, either alone or in tuberculous débris, lodging in end-arteries of the epiphysis and infecting the area supplied by the obstructed artery. The recent work of Nussbaum,[11] showing

that the arteries of the epiphysis, unlike those of the metaphysis, are not end-arteries, has cast doubt on the correctness of this theory. It is readily apparent that areas of necrosis from secondary invasion of the bone have been confused with those from primary hematogenous invasion. Some authors, as Nichols,[12] have claimed that practically all tuberculosis of the joints arises by extension from a primary focus in the adjacent bone, and they cite the presence of sequestra and necrotic bony areas as the most important evidence in favor of the contention. The mere presence, along the articular surface, of a sequestrum or of a cavity remaining after necrotic bone has been absorbed is not proof that the primary infection was in the bone. Careful examination from the standpoints of location, density, bilateral involvement and amount of sequestration will show that many of these bony lesions are the result of secondary invasion. The following is another case in point:

Male, aged sixty-eight years, had mild tuberculous arthritis of the knee for fourteen years, during which time he received no treatment and worked on the limb continuously. The symptoms then became markedly aggravated, and Fig. 15 shows the appearance of the joint in the roentgenogram eight months later. The joint was then resected, and two opposed areas of bony necrosis were found in the region of contact of mesial condyle and tuberosity, as shown in Fig. 16. That the bony invasions were approximately simultaneous and of recent date is evident from the fact that the density of the bone in the two necrotic areas and in the surrounding living bone is the same and is considerably less than normal. Gross and microscopic examinations of the regions involved showed only partial sequestration of the dead bone and an equal degree of atrophy in the dead and living bone.

In case of cold abscess and fistula formation in tuberculous joints, there may be invasion and infection by pyogenic organisms, greatly complicating the pathological picture. After pyogenic arthritis has been engrafted on tuberculous arthritis, the destructive effects of the former may be seen in the regions

of contact and pressure and all articular cartilage killed *en masse* by the tuberculous process may be rapidly destroyed. However,

FIG. 15.—Tuberculosis of knee of fourteen years' standing in a man aged sixty-eight years. Secondary kissing sequestra, seen in Fig. 16, in mesial condyle and tuberosity, not recognizable in roentgenogram because both the dead and the surrounding living bone were equally atrophic.

tuberculous joints with fistulæ of even long standing very frequently show no evidence of secondary pyogenic infection.

Bearing of Pressure on Treatment. It is readily seen from the pathological changes in articular surfaces that contact and pressure have a bearing on treatment and that it is different in pyogenic from what it is in tuberculous arthritis.

In pyogenic arthritis the aim of treatment should be to limit and overcome the infection and to preserve motion. The agencies which assist in the realization of one of these aims may be helpful, indifferent or harmful in the realization of the other. Pressure exerts an unfavorable influence on the spread of the infection, inasmuch as it disposes to invasion and destruction of articular surfaces at points of contact, and destruction of articular surfaces

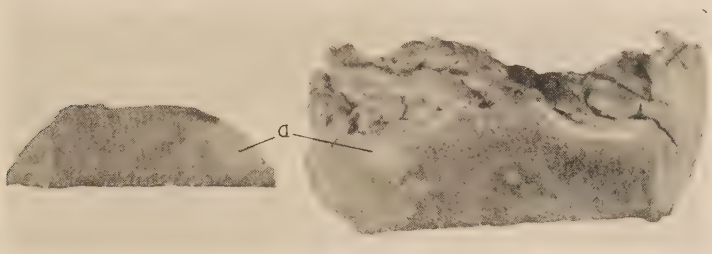

Fig. 16.—Resected specimens from joint in Fig. 15. Coronal section through tibia and sagittal section through mesial condyle, showing kissing sequestra (a) where it and mesial tuberosity were in contact. The necrotic areas of bone atrophic and partially sequestrated, showing recent development.

disposes to the development of ankylosis. Pressure is increased by weight-bearing and by marked effusion in the joint, distending the capsule and forcing articular surfaces together. Pressure may be relieved by drainage and by extension. Drainage acts beneficially by permitting the escape of noxious and of necrotic substances, to some extent by the relief of pressure. Incision should be adequate for the degree of the infection and should be resorted to oftener than is commonly practised in the milder cases. The danger of aggravating a severe turbid serous or seropurulent arthritis by arthrotomy plus careful postoperative dressings, except in the presence of a severer neighboring infection, as osteomyelitis, has been exaggerated. Limitation of

motion following loss of articular surfaces only at the points of pressure may be lessened or obviated in this way. The insertion of a drain is unnecessary where the overlying soft parts are thin, but where thick, as at the hip or shoulder, a tube should be inserted and its end sutured to the synovia. Excepting in very mild cases, pyogenic arthritis should be treated during the active stage of the disease by extension, unless it is impractical because of the location of the joint or the presence of neighboring disease. In view of the pathological changes that result from pressure, it would seem good practice to diminish or entirely relieve it by extension, unless there are very strong arguments to the contrary. The main argument to be advanced against extension is that it interferes with motion. Moving the joint acts beneficially by increasing drainage and by obviating continuous pressure of opposed articular surfaces in one region. Willems[13] has advocated motion and, when possible, weight-bearing at every stage of arthritis, because they assist in drainage. It should be remembered that, in general, motion is harmful to infected tissues and that friction and pressure of opposing articular surfaces favor cartilage destruction. Does the advantage of drainage derived from pressure by weight-bearing outweigh the advantage of protection of articular cartilage derived from extension, and is the increased drainage produced by motion more beneficial than rest? It is a curious fact that almost no mention is made by Willems of the pathological changes that may occur in the articular surfaces and of the influences they may have on the therapeutic result, particularly as concerns mobility of the joint. It is impossible to conclude from Willems' writings whether he holds that if one follows his methods there will be no pathology in the articular surfaces, or that, regardless of the extent of changes from the infection, his methods will give a better functional result than any other.

A rational management of pyogenic arthritis consists in early and free drainage and, whenever possible, extension of the joint. Mobilization with the weight lifted should be inaugurated as soon as one feels that it will be tolerated. Whether or not it

should be carried out from the beginning of treatment is a question that is still open for debate. Mobilization in arthritis is often very difficult to carry out, because of the severity of the general condition, as sepsis, or of the regional condition, as osteomyelitis, of which it is a complication. In general, mobilization should be started only after the peak of the acute infection has been passed, and extension should be continued along with it until general symptoms have partially subsided and the discharge has become slight and has lost its purulent nature. By that time the danger of augmenting by pressure the infection of articular surfaces has passed and motion for the purpose of restoring function may be pushed to the limits of toleration.

In the treatment of tuberculous arthritis there is more variation of opinion, both as to aims that should be sought and as to methods of achieving them, than is the case in the treatment of pyogenic arthritis. By operative treatment one generally aims to obtain healing with bony ankylosis, which, once established, is the surest safeguard against recurrence. By non-operative measures the majority of surgeons try to obtain healing with the greatest possible limitation of motion, preferably with ankylosis, while a few strive for healing in some cases with preservation of motion. Pressure plays a role in the non-operative treatment, in that it modifies the time and manner of cartilage destruction, and after cartilage is destroyed, it disposes to erosion or extensive invasion and necrosis of bone to which it is applied. By leading to extensive necrosis of bone followed by sequestration, it may create the necessity for operative interference in the course of conservative treatment.

Immobilization without extension of the joint in the earlier stages of tuberculous arthritis should theoretically be the best method for preservation of cartilage at the points of contact and pressure. Extension would tend to pull the surfaces apart, and in that particular would enable the granulation tissue to get at and destroy more of the articular cartilage. At the same time extension brings into play the favorable factor of fixation, which tends to lessen the amount of granulation tissue

formed. This makes it difficult to estimate the separate effects of extension and of fixation on the articular cartilage. If preservation of articular cartilage is what is desired, there are in the pathological findings certain grounds for believing that it is more likely to be realized from fixation alone than from fixation plus extension. Surely, those who argue that if extension is not applied, articular cartilage will soon be destroyed in the regions of contact and pressure are in error. There seem to be perhaps equally good grounds for arguing that if extension is applied, articular cartilage will be destroyed earlier than usual in those regions, because then the granulations would have a better chance to get at its surfaces. It is even possible that some of the beneficial effects derived from extension have come from this more rapid loss of cartilage, which puts the joint in a more favorable condition for the occurrence of ankylosis. Theoretically, it would appear that healing with motion is more apt to come from the treatment of early tuberculous arthritis by immobilization alone than by extension, because immobilization alone is more apt to preserve cartilage, and preservation of cartilage is a prerequisite for motion. Lorenz and others claimed improved results from treatment by immobilization plus weight-bearing in tuberculous arthritis of the lower extremity. If this is right, it should be worth while to investigate whether the extra pressure of weight-bearing is the beneficial factor, and, if so, whether it acts by hastening the destruction of articular cartilage or by assisting in its preservation. Once articular cartilage is destroyed in a case of tuberculous arthritis, extension would seem a rational procedure if the disease is progressive, since it should then protect the bone from erosion or extensive invasion with sequestration at the points of pressure.

Further roentgenological and clinical observations, made with the anatomical changes herein noted kept in mind, should throw additional light upon the influence which pressure has upon the therapeutic results in both pyogenic and tuberculous infections of joints. They should be made on patients treated with and without extension and with and without weight-bearing.

Conclusions. 1. In pyogenic arthritis articular cartilage is killed and broken down first at the points of contact and pressure of opposing articular surfaces.

2. In tuberculous arthritis articular cartilage is not killed first, but is protected at the points of contact and pressure of opposing articular surfaces. Cartilage is extensively destroyed first along the free surfaces, where the tuberculous granulations can grow onto and remove it. It usually disappears last in the regions of contact and greatest pressure in the joint, where it is detached and killed by undermining granulations and is then partly eroded by pressure of opposing bony surfaces.

3. Proteolytic ferments derived largely from polymorphonuclear leukocytes assist greatly in the rapid removal of necrotic cartilage in pyogenic arthritis. Proteolytic ferments are absent in tuberculous arthritis and masses of dead cartilage may persist for months or years, showing few signs of progressive destruction.

4. In pyogenic arthritis the infection rarely invades secondarily the deeper portions of the bone at the points of pressure.

5. In tuberculous arthritis invasion of the bone at the points of pressure is of common occurrence after the articular cartilage has been largely or wholly destroyed. The invasion is frequently on both sides, in which case it may lead to the formation of kissing sequestra at the points of greatest pressure in the joint.

6. Extension should be applied during the active period of pyogenic arthritis to lessen the amount of invasion and destruction of articular surfaces at the points of contact and pressure.

7. On the other hand, it would appear that extension for preventing the destruction of articular cartilage in tuberculous arthritis is ineffective. But when articular cartilage has already been destroyed, extension should lessen the tendency to erosion or invasion with sequestration of bone at the points of greatest pressure in the joint.

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DISCUSSION

Dr. W. W. Keen, Philadelphia, Pa.:

I would like to interpolate a very few remarks on one subject. Medicine is classed as one of the "learned professions." My ears were startled the other day and my mind not a little disturbed when I heard in conversation the remark that "I operated him," as if the patient had been a sewing machine or a loom. We do operate machines. We operate *on* or *upon* patients.

So far as I remember I have not heard this flagrantly bad English in a single paper in these two days, and I do not recall hearing at other meetings of the Association any such an error as this. I think it ought to be the duty of the editors of the papers presented in all of our medical societies to eliminate it. What is an editor for but for that very purpose? I am very urgent that as a profession we should use the English language forcibly and correctly.

REACTION AND REACTIONARY GROWTHS IN BONE (OSTITIS FIBROSA, ETC.)

By FREDERIC J. COTTON, M.D.
BOSTON, MASS.

This brief report results from an attempt to clear up my own very foggy ideas as to bone pathology apart from the malignant processes and those recognized as due to known infections.

The title chosen is ostitis fibrosa.

I do not believe that there is, in fact, any such disease as ostitis fibrosa, but some of my cases will have to be so entitled until we have a better term, and the histological picture called ostitis fibrosa is one that constantly recurs in other non-malignant processes.

My belief is that it is simply a stage in the sequence of processes of absorption and repair, not an independent pathological manifestation.

In attempting to attain some mental order in these matters, I have been very dependent on Dr. Frank B. Mallory, pathologist. He has examined and reported on all the specimens and interpreted them with an authority to which I cannot pretend.

Case 1.—The first case that started us was one of a diffuse, rapidly growing expansion of two-thirds of a tibia in a healthy woman of forty. Duration, six weeks. Cause, probably trauma of a fall about seven weeks before. *X*-ray might have been Ewing's tumor, syphilis or ostitis fibrosa.

Operation disclosed a spongy, uneven mass of bone, of which a great quantity was removed. Recovery uneventful, complete and without recurrence after nine months.

Histologically the picture was one of scattered, minute necroses, being bitten away by giant cells, of new bone being laid down, of extensive fibrosis in the lacunar spaces.

CASE 2.—A girl, aged eighteen years, with a like process in the neck and upper shaft of the femur, of a few months' duration, with only vague lameness. *X*-ray showed a picture of expanded growth like that of Case 1, which I diagnosed as ostitis fibrosa—probably.

Operation by Dr. Custis Hall, of Washington, D. C., showed the same diffuse, patchy sort of bone.

The specimens examined by Dr. Mallory and myself were practically identical with those of Case 1, save that the fibrosis was more intense.

In this case the causal factor seemed not to be trauma; a year before she had had a serious illness, with multiple furunculosis.

CASE 3.—Was a massive, diffuse, tumor-like growth of the greater part of a fibula. No pain, no temperature. Observed for a month it grew perceptibly (as palpated), as checked by the *x*-ray.

Operated on it showed bone much like that in the last 2 cases, not quite so patchy.

Histologically it was definite-ostitis fibrosa.

A very large amount was removed. Repair was aseptic and recovery permanent to date.

Two widely-separated pin-point abscesses were found in this case at operation. Culture from these showed pure Staphylococcus pyogenes aureus.

A year before he had had a frank localized osteomyelitis of the lower end of this fibula, long since healed, never invading the midshaft, where the "ostitis fibrosis" was.

CASE 4.—Male, aged twenty-eight years. Fibular tumor very like the last, of gradual growth for about three years. Healed fistula from gunshot wound near the ankle, a war wound received five years before.

In this case there had been occasional lameness with subjective temperature, as one sees with Brodie's abscess cases.

The *x*-ray showed no abscess, but an ostitis fibrosa picture. Here, again, operation showed a picture of patchy, rather soft overgrowth of bone.

Here, again, two pin-point abscesses, three inches apart, with positive Staphylococcus pyogenes aureus cultures, told the story.

But, save at these points, the histological finding proved to be the same ostitis fibrosa picture.

In this case, again, repair was without pus or temperature reaction, and cure seems permanent, without renewal of bone growth.

So far we have 4 cases of what will have to go, for the major portion of the involved area, as ostitis fibrosa—1 due to trauma, 1 possibly the aftermath of infection and 2 definitely infective in origin.

It lies near to suppose that we have here no one disease, but a stage in the reaction to stimuli, *infective or not*, that do not produce pus or massive bone necrosis.

Is there a sequence in which this condition is a stage?

I think there is.

Case 5.—Boy, aged eighteen years. Tumor of proximal phalanx of right mid-finger, palmar side. Duration, six months. History of the repeated normal traumata of a school ball-player. Little lameness, but growing tumor.

X-ray showed bone expansion, etc., but did not serve for any diagnosis. Clinical diagnosis, "traumatic ostitis."

Opened, this finger showed fluid, no pus, but a minute cavity in the phalanx surrounded by infiltrated bone.

Histologically, much the finding of the previous cases, but with fibrosis not yet conspicuous.

The fluid contained, surprisingly, Staphylococcus pyogenes albus in pure culture. No pus; prompt healing; no recurrence.

This was an early case, or less severe.

CASE 6.—Semilunar bone of wrist. Man, aged forty years. Lame wrist for several months. Trouble attributed to minor trauma. Type case of "traumatic osteitis of semilunar," "Preiser's disease."

Excision—healing.

Histologically, cancellous bone, rather dense, with moderate definite fibrosis.

CASE 7.—Dr. P., aged forty-five years. Suffering inconvenience rather than pain from an enormous expansion of the whole of one tibia of several years' slow growth. Repeated traumata in the saddle. *X*-rays showed picture of what we have called Paget's disease, but of one bone only. Operation disclosed irregular, but, on the whole, dense bone without marrow cavity. Of this 14 ounces, by weight, was removed. Prompt healing, early weight-bearing, no return in the four years since the operation. The specimens now reëxamined by us show histologically very dense bone alternating with ivory bone. There was here no fibrosis, almost no lacunar spaces.

The picture histologically is exactly that of a specimen for comparison from organized callus obtained at operation for vicious union a year after fracture.

It would appear from this that the picture we have called ostitis fibrosa is no more than a picture of destruction of bone, with the giant-cell osteoclasts familiar to this process, of building up of bone by normal layering-on of osteoid, both processes normally seen in growth and in repair of fractures or wounds, while the fibrosis, the conversion of granulation tissue to fibrous tissue in the open spaces, is just what one finds in callus tissue—just what I found years ago in an experimental series as a normal process in grafted cancellous bone. In fact, a "taking" graft of cancellous bone is exactly ostitis fibrosa histologically.

Moreover, in going over Dr. Mallory's slides of typical Paget's disease, this same picture of absorption and repair, of giant cells, of granulation tissue undergoing fibrosis, often intense, is what we found.

Earlier cases (Case 5) show granulation tissue not yet fibrosed; very late similar cases show dense bone without large lacunar spaces and no fibrosis. In other words, neither fibrosis nor giant cells belong to the later-picture.

Ostitis fibrosa (even with a process of absorption and of tissue growth simulating actual tumor) may be then no more than the expression of an intense reaction to an irritant.

What the irritant is in the purely traumatic cases is hard to say. Mallory's studies of the endothelial giant cell make it clear that the presence of devitalized bone (in bone or as bone dust injected into muscle) brings the giant cells that contrive the actual absorption, and it lies near to suppose that trauma may kill trabeculæ by jar, by pressure from hemorrhage or from damage to circulation. In the cases of assumed general pathology, of Paget's disease for example, circulatory changes may be the antecedent cause, with bone death, bone absorption, repair and fibrosis as the sequence.

In any case the process once started *may* perpetuate itself through excess of pressure, due to the granulation tissue causing fresh molecular death of bone, and so on.

Only in this way can one account for the effect of the operative cures, for the immediate cessation of growth previously continuous, when the growth is cut into, but only in part removed, as happened in Cases 1, 2, 3, 4 and 9.

Far more intense giant-cell action, with much new bone, alternating in patches with whole portions predominantly fibrous, were found in a second operation on the rapidly growing new bone of a tibia from which a central sequestrum had been removed by Dr. Kimpton (Case 8).

Extreme fibrosis, with multiple, tiny, actual cysts, apparently from absorption—literally lined with giant cells—characterized sections in a case (Case 9) of Dr. J. W. White's, in which three bones were involved in a process which one could only call ostitis fibrosa clinically.

Larger cysts, with giant-celled lining or filled with the giant-celled tissue which Mallory has so clearly shown to be granula-

tion tissue, with endothelial giant cells, appear in case after case of v. Recklinghausen's series of ostitis fibrosa.

Lately, I have seen a case (Case 10), with Dr. George Morse, of a very large cyst of the femur near the knee, solidly lined with giant-celled tissue, and we have all seen repeatedly the cysts that have only a fibrosed layer for lining.

Lately, I opened up one in which the remains of lining consisted largely of endothelial leukocytes crammed with fat from the degenerated tissues (Case 11).

Here, again, tension conditions play an obvious part.

In only one of my own cases have I seen local recurrence of giant-celled tissue, and this in a case of jaw "tumor," in which, after the first operation, it may well have been that tension conditions were reproduced in healing. A second clearing out gave healing, now solid after a year and a half (Case 11).

In other words, the development of the bone-eating tissue may go on extensively, but it tends to cease the moment tension is relieved.

Here, again, the recent results of *x*-ray treatment in the giant cell cases shown from Detroit and from the Memorial Hospital in New York seem significant.

X-ray therapy does not check but enormously accelerates bone destruction until the cortical layer is gone, and then rapid regeneration recurs, though with a deformity of bone (from mechanical crumpling), so in excess of what one sees after operative treatment as to make this a very poor method, in my opinion. But the point is that it cures by relief of tension.

It must be borne in mind, also, that there is an obvious tendency to cure in these cases whether from relief of tension through fracture or operation or from other cause—that neither the tumors nor cysts nor recognizable remains of them are met with in patients past thirty.

To sum up, I submit that we have every reason to think that stimuli of trauma with resultant direct damage to bone or indirect damage through hemorrhage or malnutrition may give a process exactly like that found in infections, infections not sufficient for

frank osteomyelitis—that certain systemic conditions may be in some fashion, perhaps through vascular changes or sheer malnutrition, the cause of exactly similar local changes, and that these changes result from processes of absorption, giant-celled activity, deposit of bone, fibrosis of granulation tissue, all present in normal bone repair and in no sense pathological in themselves —that the process is one of reaction, not an independent pathology, that it includes what we have called ostitis fibrosa.

It seems probable that granulation tissue tumors ("giant-celled" tumors) and bone cysts represent a local excess of a similar process, whether one accepts Barrie's classification of hemorrhagic osteomyelitis or not.

If this conception is correct, we come down to a classification, pathologically, of:

1. Neoplasms.
2. Osteomyelitis (of frankly destructive type). Characterized by *mass* necrosis with pus formation.
3. Inflammatory *reactions*, sometimes tumor-like, characterized particularly by *molecular* death of bone and absorption of this by giant cells with no differentiating line between:
   - (*a*) Inflammatory infiltration (sometimes of infectious origin).
   - (*b*) "Ostitis fibrosia."
   - (*c*) Denser bony infiltration (late repair picture).
   - (*d*) Granulation tissue masses, ("giant-celled tumor").
   - (*e*) Bone cyst.

If this proves valid it gives ground for coöperation between the surgeon and the pathologist of whom we have expected impossibilities. Also, it may give a point of departure for clearer *x*-ray interpretation if we can group as apart from the neoplasms a group of "reactionary processes and reactionary tumors in bone."

THE TREATMENT OF COLLES' FRACTURE[1]

By WILLIAM DARRACH, A.M., M.D.
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It is not intended to present an exhaustive study of this subject, but to call attention to certain points thought to be of importance.

The term Colles' fracture, as used today, includes those fractures of the radius which involve the cancellous portion of the lower extremity of that bone. They are usually due to indirect violence, the force being transmitted through the palm of the hand. The mechanism is unimportant.

Our main theme is that we should base our treatment of these fractures on a careful study of the pathology of each individual case, rather than blindly follow a set procedure in all instances. Detailed examination of the *x*-ray and clinical evidence, before reduction, if practicable, should determine the course to be pursued.

Pathology of the Fracture Itself. The plane of fracture may be horizontal or oblique. The plane of fracture may be single or multiple. There is usually considerable fragmentation, especially of the dorsal surface.

The following *displacements* may be encountered, either alone or in combination. The lower fragment may be *shifted*, *i. e.*, moved without changing the direction of its vertical axis; or it may be *tilted*, so that its vertical axis makes an angle with that of

[1] This paper was presented before the New York Committee, appointed by the General Fracture Committee of the American College of Surgeons. The discussion is embodied in the paper. It may therefore be considered as an official report from them.

the upper fragment (angular deformity); there may be a proximal displacement, either *impaction* or *overriding;* there may be a rotation on the vertical axis:

Shifting: Radial.
Ulnar.
Dorsal.
Ventral.
Tilting: Radial.
Ulnar.
Dorsal.
Ventral.
Impaction.
Overriding.

Rotation, clockwise and counterclockwise. The typical deformity found in the classic case is a combination of radial and dorsal shifting, dorsal tilting and impaction. But there are many cases which do not present this condition, and the variety of possible combinations is very large.

Of the Associated Injuries. The injuries to tissues other than the bone, associated with this lesion, must be considered in connection with outlining a plan of treatment. The extent of laceration of the pronator quadratus, tendons of both dorsal and ventral groups, their sheaths, involvement of the median and ulnar nerves, all demand attention. The inferior radio-ulnar joint is a very important factor. Its articular surfaces are frequently disarranged by the displacement of the lower radial fragment. Moreover, its ligaments are very apt to be torn or stretched. Anterior dislocation of the ulnar head is far from rare, especially with marked dorsal displacement of the lower radial fragment. The ulnar styloid, or its attached internal lateral ligament, is often torn away. Occasionally an associated fracture of the carpus is found.

TREATMENT. *Reduction of Displacement.* This is not always necessary. There may be no displacement. Again, it may be so slight or of such little consequence that it can be allowed to remain. In certain comminuted cases it may be wiser not to

attempt correction of moderate displacements lest the necessary manipulations make things worse. In most cases with displacement, however, an attempt should be made as early as possible, and almost always under anesthesia.

The manipulations should be planned out according to the character of the displacement. The method usually recommended is to use traction with hyperextension, rocking of the lower fragment to break up impaction, followed by flexion, with pressure directly over the fragments. In the typical deformity this method will generally be successful, but the hyperextension may do more harm than good by adding to the comminution or by added laceration of the soft parts. We should combine *traction* to disengage the fragments, *movements* at the radiocarpal joint to overcome shifting or tilting, aided by direct pressure over the upper and lower fragments. Rocking the lower fragment back and forth to loosen impaction should be done gently, only using the extreme positions when necessary.

The late disabilities associated with each type of displacement should be considered in order to determine how far the attempted reduction should be carried and how often repeated. *Ventral* or *dorsal shifting* of a moderate degree leads to slight if any impairment. If marked, it affects the radioulnar joint and the latter shares in the silver-fork deformity. When extreme, dorsal shifting may interfere with flexion of the fingers, owing to the sharp anterior lower edge of the upper fragment impinging against the tendons just before they enter the carpal canal. Occasionally it will press on the median nerve. *Ulnar* or *radial shifting* tends to interfere with the radioulnar joint, and so with pronation and supination. Radial shifting leads to prominence of the ulnar head.

Ventral tilting is of less serious consequence than commoner *dorsal tilting*. Both of these change the arc of motion of flexion and extension at the wrist and the latter exaggerates the silver-fork deformity. Dorsal tilting should be overcome whenever possible. The rarer forms of *radial* or *ulnar tilting*, *i. e.*, rotation of the lower fragment around an anteroposterior axis, change

the arc of motion in adduction and abduction, and may also derange the radioulnar joint.

Impaction is of the greatest importance. Its reduction is difficult both to obtain and to maintain. It emphasizes the ulnar head prominence and interferes with the radioulnar joint. Any lesion which shortens the radius without shortening the ulna throws out of alignment this joint, interfering with pronation and supination and leading to pain referred to the region, which may persist for years.

Overriding is rare. When it does occur there is so much laceration of the soft parts that reduction is rather easily accomplished if the attempt is made early.

Rotation of the lower fragment around a vertical axis is uncommon. It is rather difficult to establish and correct.

Comminution or *multiple planes* complicate the procedure. Manipulation aimed to bring one fragment into position will often force another into faulty alignment. Traction and pressure are often the only procedures indicated.

There is one type which deserves special attention, which has been called "*dorsal comminution*" or "loose *dorsal fragment.*" The lateral *x*-ray will sometimes show the ventral surface of the radius intact except for a single plane of fracture, but on the dorsal aspect several small fragments or one of considerable size, lying almost horizontal. These are usually associated with marked dorsal tilting. They will be referred to later in connection with the case where the plane of fracture is oblique instead of horizontal.

All of these factors should be considered in deciding whether a reduction should be attempted, how it should be done, and in case of partial or complete failure, how many further attempts should be made. We have on several occasions made four separate attempts.

Maintaining Reduction. After the fragments have been brought into as favorable alignment as possible, the next problem is to maintain that position. The simple transverse fracture, without comminution, will stand a good deal of manipulation

without slipping out of place. The cases which originally presented impaction, especially those with dorsal tilting, are more liable to recurrence of displacement. A very disappointing group are those with dorsal comminution or with an oblique plane of fracture. After the reduction of any Colles', and especially after recovery from the anesthetic, the lower fragment is being constantly pulled against the upper by the forearm muscles. This is of great advantage in the horizontal simple cases, but in the oblique cases the lower fragment tends to slide upward, and either forward or backward, according to the plane of obliquity. In the dorsal comminution cases the upward pressure on the lower fragment is opposed in front by the contact of the two otherwise intact cortical shells. Behind, however, there is less opposition and the lower fragment tends to slowly but steadily tilt backward. It is a depressing sensation after having seen a very satisfactory postreduction *x*-ray to find a week or ten days later that the displacement has recurred. A few embarrassing experiences of this kind, together with an observation made during an open reduction, led us to believe that if the wrist were kept in a flexed position the danger of recurrence was less. After several years' trial this has been used as routine in all cases of this kind, and the results warrant its being continued.

The type of splint used is not of great importance, provided it gives efficient and comfortable immobilization and permits of ready access to the part. Our preference is for two molded plaster splints, not too tightly applied. The palmar splint should not extend beyond the midpalmar fold nor high enough to impede movements at the elbow. In the few extreme cases, where it is desirable to limit pronation and supination, the splint must go a short distance above the flexed elbow. Thumb and fingers are to be left free. The wrist should be in a position of rest unless flexion is indicated.

Massage and Movements. If the wide variability in the pathology as described above is considered, it becomes evident that no set date can be set down for beginning massage or movements. The decision must be made in each case according to the factors

involved. In some cases, with no displacement or with a complete reduction of a single horizontal displacement, massage and movements can be started the same day. In the cases needing the flexed position these should be delayed for from eighteen to twenty-one days. The main question is, How soon will union be firm enough to prevent recurrence of the displacement. But we must also bear in mind the danger of too prolonged immobilization. In some of the badly comminuted cases where a good reduction cannot be obtained, movements should be started long before union is firm. This is especially true in older people where function is so difficult to obtain and where deformity may not be so important.

Massage and movements during the early period must be done by the surgeon himself or a well-trained assistant. They had better be delayed than trusted to unskilled hands. Passive motion should be very gentle and used more as a guide for active motion. If carried beyond the point of pain it is capable of considerable harm.

Operative Treatment. Cases which present themselves after firm union has taken place, complaining of marked disability or persistent pain, can often be helped a good deal. In some an *osteotomy*, simple or cuneiform, may be indicated. The results here are not always brilliant. It is difficult to maintain the position of the lower fragment, and the radius is usually shortened, with the resultant derangement of the radioulnar joint.

Where there is considerable interference with pronation and supination, with pain referred to the region of the ulnar head, much relief can be obtained by a *subperiosteal resection of the ulnar head*. This will also relieve the deformity due to the prominence of the latter. It is a procedure which has a rapid convalescence, as no splinting is necessary. It should be noted here that the degree of disability does not correspond to deformity. In some of the most exaggerated cases of relatively lowered ulnar head, there is little or no disability or pain. This may be due to the complete rupture of the ligaments, so that the ulnar head has free play and the joint surfaces are not in contact at all.

Another group can be materially helped by a different non-reducing operation. Where there has been marked dorsal shifting and the sharp edge of the upper fragment is interfering with the flexor tendons, an osteotomy of the projecting lip will give marked and rapid relief. It may be necessary to combine these two procedures on the same case.

Open reduction in early cases is rarely indicated. It may be necessary to remove loose fragments that are causing difficulty and cannot be replaced.

SUMMARY. 1. Early reduction based on a careful study of the details of the displacement.

2. The flexed position for certain cases.

3. The marked variation in the liability to recurrence of the displacement.

4. Non-reducing operations for late cases.

THE FATE OF THE FRACTURED CARPAL NAVICULAR

By KELLOGG SPEED, M.D.
CHICAGO, ILL.

The position of the navicular bone in the carpus is probably one cause of its injury in falls on the hand or similar traumata, such as backfire jerks when cranking an automobile engine. Although the bone lies in and dominates the proximal row of the carpus, its position changes over a wider arc in all wrist movements than any other carpal bone. This bone attempts also to ally itself with the distal row. If falls are sustained on a hand in true adduction, the navicular bears much of the brunt of the forced extension of the wrist and the conveyance of force to the forearm. Navicular fracture, with or without dislocation of one or both fragments, is a fairly frequent occurrence.

The blood supply of all the carpal bones is rather scant, arising from terminal branches of the radial and ulnar arteries, which spread out in a periosteal branching without the formation of a true nutrient artery. Some blood reaches the bone via the ligamentous attachments. The navicular receives its supply from small vessels, which enter about the middle of the bone and spread out as indicated in the periosteum, to supply both the cartilage and osseous tissue. As the usual site of fracture of this bone is near its middle, because of its peculiar anatomical position, the blood supply is always seriously interfered with immediately, inasmuch as these small vessels are torn off by the trauma. Even the ligamentous supply can be cut off by lacerations.

Men sustaining carpal injuries may not present gross deformities, crepitus or much immediate change in function, so that a large proportion of the fractures are overlooked clinically. It is

also true that even when a skiagram is made of the wrist a fracture may not be noticed. There results, consequently, nonrecognition of the fracture of the navicular; advice is given to use the wrist. Baking, forced passive movements and active movements are insisted upon, and no fracture diagnosis is made until weeks, months or years later, when the dysfunction becomes so prominent that someone interprets the condition correctly from the symptoms and a skiagram. In some patients not only is the navicular fractured, but one of the fragments, usually the proximal, is dislocated out of position in relation to the radius and the surrounding carpal bones. This fragment dislocation may be accompanied by its companion in the proximal carpal row, the lunate. It is not my purpose to go into the symptomatology, but to attempt a description of the fate of the bone.

The course of the life of the navicular after a fracture may take either of two different paths. Fracture without separation (dislocation) of fragment or with impaction, which is recognized and treated by early and prolonged immobilization, may heal satisfactorily without untoward results. This fact has been shown clinically.

The fracture unrecognized or discovered after a lapse of a week or more, during which motion and use have been advised, pursues a different course. It is with this quite common result that we must deal. After the unrecognized and misused fracture, pain increases for a period of several years. Along with the pain comes diminution of functional use of the wrist, hand and forearm, betrayed by lack of hand grip, decreased wrist motions and atrophy of the forearm muscles.

In the navicular bone after complete fracture, excepting isolated fractures of the tuberosity, blood supply is rapidly cut off and the main mass of the bone cells undergoes slow death. Just how long it takes for this death to occur cannot be positively stated; probably it is complete within a week. The navicular then at first retains its original density, and as the weeks and months pass, when pain inhibits use of the other bones of the hand and forearm, there is seen a marked contrast between the

navicular and neighboring bones, which must lose calcium in the process of atrophy of disuse. This atrophy can be demonstrated even in the forearm bones. Formation of a new blood supply is constantly discouraged by movements of use which overcome the delicate efforts of the small vessels involved. As attempts at use or passive movements are continued, the loss of blood supply of the bone becomes permanent.

A few weeks after injury a skiagram shows the plane of fracture, a navicular uniformly dense and with deeper shadow than the other carpal bones. As time passes the plane of fracture appears to widen and less difficulty is experienced in making a roentgenologic diagnosis. Eventually the bone appears to undergo absorption in its body. The cartilaginous and cortical edges retain their outline markings for many months, but in three to five years light spots appear in the bone shadow quite remote from and not necessarily connected with the apparent bone absorption going on at the fracture plane. These lighter areas may be puzzling unless one studies the pathology minutely. New-bone formation from surrounding bones begins to appear in one to two years, and if an average of five years is taken, we may expect to find considerable exostosis formation manifested from the lower articular end of the radius. The degree of this new-bone development is probably governed by the amount of forced work attempted by the injured hand. In women who do little manual labor the exostoses are minimal; in men the amount of bone thrown out may cloud the lines of the wrist and carpus and appear to be sufficient to cause bony ankylosis.

At operation on the fractured navicular three to five years after the break, when the carpal joint is opened synovial fluid is present. This is not abnormal, except that it may be slightly thicker and stained a deeper yellow color than normal. The bone when exposed to view seems eroded, its surface may present pits and the cartilage may be wanting, especially near the fracture plane. On the whole, however, the cartilage retains its color, apparently is living, and may have its normal smooth or shiny surface. When an attempt is made to remove the bone fragments, they are

found to be quite friable, and if care is taken to remove them *en masse*, one finds that the bone has become a mere shell of surrounding cartilage and cortex which encloses a yellow, fat-like material. This internal portion of the bone is all that remains of the former rather dense cancellous tissue of the body of the navicular. It may easily be scraped and lifted out from the enclosing shell. There is erosion and absorption of bone grossly evident at the plane of fracture, and although only two fragments could be made out in the skiagram, three or more may be found in the specimen. Overlying shreds of old capsular ligament may be adherent to the bone, especially at the tubercle. As a rule, the joint capsule has completely healed and is represented by an abnormally thick, whitish, fibrous material.

Specimens of the untreated fractured navicular removed within six months after the time of injury show on microscopic examination that the main mass of the cancellous bone tissue is dead. No normally staining nuclei can be found in the bone cells. At the fracture surface two general changes can be noted, one regenerative and the other degenerative. There is a very faint effect at new bone or callus formation. This is really only in microscopic areas. The main change is degenerative. The mass of cancellous tissue near the fracture plane has lost its cellular identity. Extending from the fracture surface is an invasion of small round cells in which are scattered many of the osteoclastic giant multinuclear cells marking a steady progressive absorption of the bone. The cortex and cartilaginous rim usually present a better resistance, and around the edges of the bone the section shows cells and nuclei with practically normal staining reaction, evidently still alive. The main mass of the cancellous tissue, however, is dead.

After four or five years the whole interior of the bone is soft and fat-like. On section of this material nothing is found but fibrous tissue—no fat, no trace of bone cells or lamellæ. Around the bone edge the shell is composed of cortical bone with a few rows of bone cells containing nuclei and with fairly healthful cartilage covering where it should be present.

After a study of a series of these fractures at varying periods after the original trauma, I feel justified in saying that the original fracture interference with the blood supply cuts off nutrition to the cancellous structure. Should the condition be recognized and sufficient rest be offered the bone, revascularization with a saving of the bone may result. In the neglected cases bone death rapidly ensues, and the process of malacia or osteoporosis begins, ending in complete absorption of the main mass of the bone. As far as I can determine we must revise our ideas about the influence of the synovial fluid on these fractures. It has formerly been taught that the irritative effect of the synovial fluid led to destruction of the bone, but my sections seem to prove just the opposite, namely, that the synovial fluid offers sustenance to the superficial layer of cartilage and cortical bone and maintains the nutrition of the cells for years.

The new-bone formation from surrounding bones must be a reaction to the presence of the dead navicular, and is protective, that is, it is a natural effort to stiffen the wrist to inhibit motion and consequently relieve the patient from pain.

Using the clinical and pathologic evidence obtained from a series of these injuries, there is no hesitation in advising immediate and prolonged splinting of the fractured and undislocated navicular. If dislocation of fragment has occurred, I believe operative removal is indicated. Certainly, in all fractures unrecognized more than a few days operative removal of the whole bone promises the quickest return of function in the hand and wrist. Delay gains nothing.

DISCUSSION ON THE PAPERS OF DRS. DARRACH AND SPEED

Dr. Frederic J. Cotton, Boston, Mass.:

In regard to Dr. Darrach's paper, there is one thing that he did not express, and which I think is important, and that is the question of the frequency and importance of the ulnar lesion.

I happen to have seen a good many such cases that were sent to me. They could not work after the ordinary fracture.

There have been two causes outside of the arthritis cases that have occurred to give this disability: One, in which he has spoken of the long ulna which has to be dealt with, and the other, much more common, is the disability from the loose ulna head. I believe in every Colles' fracture there is not only tearing of the ulnar ligament, but considerable displacement of the ulnar head. It is very easy to overlook this. In the late cases, where the disability is very serious, if you have the patient hold his arm up, flexed, knuckles forward, you will be more apt to detect it. In quite a number of the cases that trouble has been extreme.

There are two considerations that come out of this: In the first place, the amount of repair which can be accomplished in almost all of the cases by an osteotomy of the radius and a correction of the backward displacement which brings the ulna back into pretty decent approximation to the articular surface where it belongs. This works out very well. The other thing is, we want to have a reduction which will prevent this sort of thing happening. I have found that in almost all the Colles' fractures there is a certain twisting backward of the lower fragment. I have seen case after case. They are supposed to be reduced, or reduced with the exception of that twisting, and that is the reason why I have practised and urged for a good many years the form of reduction by pronation and flexion as a routine not only in the extreme cases, but as a routine for all ordinary Colles' fractures that have to be reduced. The flexed position will prevent recurrence of dislocation as nothing else will, and recurrences of displacement are very common.

One other point: All of this classification of lesions is, of course, accurate from the surgical point of view, but we must remember that it is the practitioner who treats these cases and not the surgeon. What we want is some form of reduction which is not difficult and which will apply to all or most all of the cases. I think the form I have spoken of will take care of all cases except the anterior displacement.

I do not think that I have seen more than two or three anterior displacements. These are extremely rare. The type of reduction that I have spoken of and the routine of flexion in plaster for a couple of weeks will give you better results, and can be carried out by any man with a pair of good, strong hands.

As to Dr. Speed's paper, I would like to adopt part of it. I think the process he has spoken of is exactly what I have tried to describe, and is just the same process in these isolated carpal bones as the osteitis fibrosa occurring at other places, often of traumatic origin.

Dr. E. A. Codman, Boston, Mass.:

The proper treatment of Colles' fracture seems to me a matter of organization. In our hospitals there are men who know how to set Colles' fracture. They are pretty intelligent men and they always move up in the hospital to higher things, and it remains in the out-patient department for young men to get their experience by setting them wrong. That is one of the reasons why in industrial work one sees so many badly set Colles' fractures. I think if we attended to the organization of our out-patient departments it would be far more important than going over the theories of special treatments of such lesions as Colles' fractures in the surgical societies, year after year.

In the matter of the fractured scaphoid, I believe I was the first one to recommend removal of the proximal fragment in these cases twenty odd years ago, and I have always found this a satisfactory operation until the very last case, which had a rather poor result, and the patient was only slightly benefited. In this case I removed only half of the proximal fragment. I think it is necessary to take all of the proximal fragment out, but I think it is wiser to have the distal fragment to prevent a shifting of the whole hand toward the radial side.

Dr. John H. Gibbon, Philadelphia, Pa.:

Every surgeon thinks that he knows more about Colles' fracture than anyone else, and the introduction of the subject, like appendicitis, is sure to produce discussion.

The subject of fractures certainly needs to be given more consideration in the undergraduate curriculum, and interns should not be allowed to carry out the treatment of these cases without direction.

Dr. Darrach's illustrations go a long way in explaining some of the subsequent trouble which patients with Colles' fracture have.

One of the simplest and most satisfactory methods of reducing a Colles' fracture, and one which requires no anesthetic, is that which

was used by Dr. T. S. K. Morton, a late Fellow of this Association. The method consists in interlacing the fingers of the two hands and placing them under the proximal fragment and the two thumbs on top of the distal fragment; then by a twist of the wrists, downward pressure is made on the distal fragment and upward pressure on the proximal one. It is remarkable how much force can be exerted in this way and how quickly the displacement is reduced.

In my judgment plaster should have no place in the treatment of Colles' fracture.

The important thing in regard to the fixation is that the hand should be carried to the ulnar side and flexed. Passive and active movements of the fingers and wrist should be carried out from the very first. The method of treating these fractures after reduction, with a simple sling passing around the wrist and allowing the hand to hang free in flexion and abduction, as practised by some English surgeons, has always appealed very strongly to me, and if I should be so unfortunate as to have a Colles' fracture, it is the treatment which I should employ. Complete reduction and early and complete movement of the fingers are the two most important things in the treatment of this very common fracture.

Dr. Emmet Rixford, San Francisco, Calif.:

In Cooper College, San Francisco, we early had impressed upon us the seriousness of Colles' fracture. Having in mind the number of malpractice suits which resulted from the frequently poor results of the treatment of Colles' fracture, our venerable chief, the late Dr. L. C. Lane, a Fellow of this Association, would not allow us youngsters to treat a case of Colles' fracture in the clinic.

In 1876, when Dr. Lane was in Europe, he went to Ireland to worship at the shrine of Abraham Colles. He found Colles' son a practising physician who understood that his father had done something about the broken wrist, but that he, the son, knew very little about it. Dr. Lane was grievously disappointed, but perhaps he misunderstood British reluctance to show personal feeling.

Naturally, one agrees with most of what has been said today of Colles' fracture, but I question the wisdom of advising the every-day practitioner, who treats the occasional case of Colles' fracture, to use plaster of Paris as a dressing, for we have met disaster with its use and are continually seeing bad results of its use in the hands of others. There are inherent disadvantages in the plaster splint, not the least of which is the tendency of the surgeon to leave it on too long. We believe there is great virtue in removing fracture dressings

frequently, and in the case of Colles' fracture every two or three days, for massage and manipulation.

One important little thing we learned from the late Sir William Macewen, of Glasgow, that one cannot hold the upper fragment of the radius without giving it some special support. A long volar splint presses upon the expanded lower end of the radius and tends to maintain posterior displacement of the lower fragment. A short volar splint presses upon the front of the wrist on the tendons and the median nerve and fails utterly to support the proximal fragment of the radius. Macewen used a molded volar splint with an elevation between $\frac{1}{4}$ and $\frac{1}{2}$ inches high, perhaps made of a portion of a broom-handle, so fitted as to support the proximal fragment of the radius, and therefore well above the expanded lower extremity.

We prefer to dress Colles' fracture with a long posterior and short anterior splint, with the elevation above described; for the section of broom-handle there may be substituted a roll of surgeon's lint covered with adhesive plaster, gum side out, which when placed on the forearm to the radial side of the tendons maintains its position. This apparatus, bandaging the hand in moderate flexion, as advised by Dr. Cotton, holds the fractured radius in the majority of cases most satisfactorily.

Dr. John H. Jopson, Philadelphia, Pa.:

With reference to Dr. Darrach's paper, I wish to show a few slides illustrating a method that I have used in connection with the reduction of Colles' fracture.

With regard to Dr. Gibbon's method of reduction, I have used a method illustrated by these three diagrams. The only thing about it which I stress is the use of a fulcrum. I believe it is necessary to use force in many cases. With all due deference to Dr. Darrrach and Dr. Gibbon, I believe that many of these fractures are not capable of reduction by manipulation alone, but that we have to mold them into place, and preferably with an anesthetic.

In regard to the position of the hand afterward, I do not agree with Dr. Gibbon in not using a splint, but only a sling, and I believe in the use of a splint for at least two or three weeks, and that in this way only can we prevent recurrence of deformity.

Dr. Lewis S. Pilcher, Brooklyn:

I would like to add a word to what has already been said. It has been exceedingly interesting to find out how, in a cycle of years, the same thing comes up again and again. It was fully fifty years ago

that this subject began to interest me, and I began to study the subject. In 1917 I returned to it, and with the added light that the roentgen ray had shed on it, felt that a very positive and comprehensive degree of knowledge as to this injury had been secured.

Two points, however, I would like to add to the discussion of this afternoon: As to the position of the fragments: In my earlier observations and experiments, I observed that the posterior periosteum, instead of being torn through and through, was often lifted up by the lower fragment; this lower fragment, as it was pressed backward and twisted upward, lifting up the dorsal periosteum with it. As this lower fragment became entangled upon the lower end of the upper fragment and was locked into malposition on flexion, this untorn periosteal portion remained as a strong band, which held the lower fragment in its displacement, so that this was a very common agent in resisting the reposition of the fragments into their proper relations.

Now, if one relaxes this locking band by putting the hand and wrist in the position of marked extension at the time of trying to replace the fragments, this band would be relaxed and it will be comparatively easy to replace the fragments in their proper relations.

The second point is with reference to the tendency of the lower end of the ulna to slip away from its anchorage to the radius. When the lower end of the radius is brought back into place the lower end of the ulna comes up into proper relation, but requires support to hold it there. It has been my practice for these many years to make a little roll of bandage and put it in the shallow sulcus alongside the ulna, there to be supported by proper adjustment of the later dressings. Such device should not be omitted, to counteract the tendency to lateral spreading which the pressure of simple anterior and posterior splints make.

Dr. Darrach (in closing):

One word about Dr. Speed's paper. I am sorry he did not call specific attention to what he evidently realized, namely, the association of lesions in two of the views that he showed. In both it is very distinct that the semilunar had disengaged from the head of the os magnum and was dislocated in connection with a fragment of the fractured scaphoid. This is also seen in the pictures of the fragments which he had removed. In the displacement of the proximal fragment of the scaphoid there is a clear indication for removal of that bone, as well as the semilunar if it is also displaced. The latter is of frequent occurrence.

One point which he mentioned should be emphasized, and that is

the long period of immobilization required in these cases. Frequently the wrist and the four fingers are immobilized, but the thumb is too often allowed free motion. Unless the thumb is immobilized, as well as the rest of the hand, we do not get proper immobilization of the two fragments.

In regard to my own paper, this was not an attempt to classify the various types of Colles' fractures; it was rather an attempt to classify various displacements which occur in Colles' fracture, in order to base our manipulations on a sound conception of the actual conditions in each case.

I cannot visualize Dr. Jopson's reduction, even over a padded fulcrum, without a shudder. To think of the sharp, jagged edges of the upper fragment being jammed against the tendons, tendon sheaths, radial and ulnar nerves, adding thereby to the injury, prolonging the convalescence and increasing the disability, is not pleasant. That is an example of what should *not* be done in handling Colles' fracture. I believe that gentle methods of reduction and gentle methods of examination are of tremendous importance. Dr. Pilcher's method is an added reason for the ulnar reduction.

Dr. Speed (in closing):

I know Dr. Codman's article, published in 1904, very well, and since then there has been no better American article written on the subject.

There are some twenty odd different lesions involving the carpal bones, but I merely wanted to describe the fate of these fractured naviculars.

THE INCIDENCE OF CONGENITAL CLEFTS OF THE LIP AND PALATE

By JOHN STAIGE DAVIS, M.D.
BALTIMORE, MD.

I HAVE frequently been asked how often congenital clefts of the lip and palate occur, and have never been able to give a definite answer. The object of this paper is to determine, as definitely as may be, the incidence of these clefts, and it is based on a study of the cases of congenital clefts of the lip and palate which have occurred up to March 1, 1924, in 24,158 deliveries in the obstetrical service of the Johns Hopkins Hospital[1] and in 3927 deliveries in the Hospital for the Women of Maryland.

A review of the literature shows that Froebelius, in 1865, published a report based on the number of congenital clefts of the lip and palate found among 180,000 children admitted to the St. Petersburg Foundling Hospital between the years 1833 and 1863.

He estimated that these clefts occurred once in 2400 births, and his figures have been generally accepted and constantly quoted. For a number of reasons, to be mentioned later, it is obvious that congenital clefts would occur more frequently in 180,000 total births than in the same number of admissions to any institution. Nothing further has been written on the subject.

One cannot determine the incidence of congenital clefts of the lip and palate from the records of any surgical clinic, as many children with these malformations die before they are presented

[1] I wish to thank Dr. J. Whitridge Williams, who very kindly placed at my disposal the records of the obstetrical department of the Johns Hopkins Hospital.

for operation or admission; many of the simpler cases are operated on at home, and a considerable number are never brought for operation.

The number of these cases applying for operation at the larger surgical clinics varies according to the increase in the population of the district from which that clinic draws; according to the reputation of that particular clinic for success with these cases; according to the knowledge of the public at large that much can be done for the relief of these deformities and the consequent willingness of parents to bring children for operation.

It is possible that the frequency of occurrence of congenital clefts of the lip and palate may vary in different parts of the world. This may even be true in different sections of our own country, and also there may be a difference in rural and urban communities. This seems to be indicated by the statistics compiled from the draft records of the defects found in the first 2,500,000 men examined for the United States Army in the World War, which show that in Vermont, at one extreme, the ratio of the occurrence of congenital clefts of the lip and palate per 1000 men examined was 1.55, while at the other end of the list stood Arkansas, with a ratio of 0.16 per 1000. These statistics also showed that the incidence was highest per 1000 in the agricultural sections of the northern states.

As these figures are based entirely on the examination of men of military age, no definite conclusion can be drawn from them as to the frequency of occurrence of congenital clefts, as those in females, those dying in infancy of malnutrition or following operation, and those operated on successfully are not included.

As far as I have been able to ascertain, no mention is made of congenital clefts of the lip and palate in the official birth records now in use in this country or abroad, and in consequence accurate information cannot be obtained for the whole country, or even for individual states, until uniform birth statistics are required which will cover this point.

It has been said that the frequency of occurrence of the types of congenital clefts may change, and this was noted by Bruns,

who quotes Langenbeck as saying, in 1828, "In the past eight years the simple harelip has become a rare occurrence and the cleft palate a frequent phenomenon." He also quotes Walter, in 1834, who says, "Double and complicated harelips occur at present more frequently than simple harelip, and much more frequently than thirty years ago." Bruns, in 1873, confirms this idea from his own statistics. These observations may or may not be of value, as the differences might have been caused by the fact that only the more difficult cases were brought into these particular surgical clinics.

In this study our interest is solely in the number of congenital clefts of the lip and palate which came to delivery, and not in those which are found so frequently in pathological embryos aborted in the early months of gestation.

For convenience and for purposes of comparison, I have separated the cases studied into three series.

Series A. The negro cases from the obstetrical service of the Johns Hopkins Hospital.

Series B. The white cases from the same clinic.

Series C. The white cases from the obstetrical service of the Hospital for the Women of Maryland.

SERIES A. NEGRO CASES FROM THE OBSTETRICAL DEPARTMENT, JOHNS HOPKINS HOSPITAL

Number of Congenital Clefts of the Lip and Palate in 12,520 deliveries, 7.

Nationality of Mothers: United States, 7.

Ages of Mothers: Youngest, twenty-one; oldest, thirty-six; average, 26.

Health of Mothers: Good, 6; excessive vomiting (early months), 1.

Mentality of Mothers: Usual ward type.

Ages, Health and Mentality of Fathers: No routine note.

Social Status: Ordinary ward type.

Primiparæ, 2; *multiparæ*, 5. Both of the primiparæ were twenty-one years old. Of the multipara, 1 child was the third of a twenty-four-year-old mother; 1 was the fourth of a twenty-six-year-old mother; 1 was the fourth of a thirty-year old mother; 1 was the fifth of a thirty-year-old mother and 1 was the tenth of a thirty-six-year-old mother.

Sex of Child: Male, 3; female, 4.

Year of Birth: 1899, 1; 1902, 1; 1906, 1; 1907, 1; 1915, 1; 1920, 1; 1922, 1.

Legitimate, 6; *illegitimate*, 1.

Presentation: L. O. A., 3; R. O. P., 3; R. O. A., 1.

Delivery: At term, 5. Spontaneous, 4; version, 1. Premature, 2. Both Spontaneous: seven and a half months' macerated fetus, 1; eight and a half months' child, died immediately after birth, 1.

Weight at Birth: Heaviest, 3317 gm.; lightest, 2268 gm.; average, 2648 gm.

Length at Birth: Longest, 53 cm.; shortest, 42 cm.; average, 47.7 cm.

Malformations:[1] *Alveolar Cleft Lip*, 2 cases. In 1 female child there was a right unilateral complete cleft of the lip with notching of the alveolar process on the right side. Palate intact. In 1 male child there was a bilateral complete cleft of the lip with notching of the alveolar process on both sides. Palate intact.

Alveolar Cleft Lip and Palate, 4 cases. In 2 males and 2 females there were bilateral complete clefts of the lip, alveolar process and palate.

Alveolar Cleft Palate, 1 case. In a female child there was a unilateral (side not given) complete cleft of the alveolar process and palate. Lip intact.

[1] In this paper the classification of Davis and Ritchie will be used.

Group I. *Prealveolar* (process) *cleft.* (Lip cleft; alveolar process normal.) Cleft of the palate may be associated with this group.

Group II. *Postalveolar* (process) *cleft.* (Palate cleft; alveolar process normal.)

Cleft of the lip may be associated with this group.

Group III. *Alveolar* (process) *cleft.* (Cleft follows incisor sutures.) Clefts of the lip and palate are usually associated in this group.

Associated Anomalies, 3 cases. In one girl there was clubbing of feet and hands with polydactylism. (Died on fortieth day.) In one girl there was malformation of the mandible and enlarged thymus. In one boy there was rudimentary hand and forearm and ankylosed elbow (seven and a half months' fetus, stillborn).

Mortality: 5 cases. One seven and a half months' macerated male fetus, placenta luetic; 1 eight and a half months' female child, died after a few gasps, typically luetic, placenta negative; 1 female child stillborn at term, placenta luetic; 1 male child died third day (mother had condylomata and the placenta was luetic); 1 female child died on fortieth day of inanition. Three of these were typically syphilitic in appearance, although the Wassermann reaction was negative for the mother in 1, for the mother and father in 1, and for the mother, father and cord in 1.

SERIES B. WHITE CASES FROM THE OBSTETRICAL DEPARTMENT, JOHNS HOPKINS HOSPITAL

Number of Congenital Clefts of the Lip and Palate in 11,638 deliveries, 13.

Nationality of Mothers: Bohèmian, 1; Irish, 1; Roumanian, 1; Russian, 2; United States, 8.

Ages of Mothers: Youngest, 17; oldest, 38; average, 25½.

Health of Mothers: Good, 10; excessive vomiting (in early months), 1; health poor, 1; tuberculosis of lungs, 1.

Mentality of Mothers: Ordinary ward type, 12; feeble-minded, 1.

Ages, Health and Mentality of Fathers: No routine note.

Social Status: That of ordinary ward patient.

Primiparæ, 7; *multiparæ*, 6. The ages of the primipara were 17, 19, 20, 22, 24, 26 and 33 years, and 4 of the 7 children were illegitimate. Of the multiparæ, 1 child was the second of a twenty-year-old mother; 1 was the second of a twenty-three-year-old mother; 1 was the second of a twenty-five-year-old mother; 1 was the third of a thirty-five-year-old mother; 1 was the third

of a thirty-eight-year-old mother and 1 was the sixth of a thirty-year-old mother. One of these children was illegitimate.

Sex of Child: Males, 10; females, 3.

Year of Birth: 1905, 2; 1907, 1; 1909, 1; 1911, 2; 1915, 2; 1916, 1; 1919, 2; 1921, 2.

Legitimate, 8; *illegitimate*, 5.

Presentation: L. O. A., 7; L. O. T., 1; R. O. A., 4; R. O. P., 1.

Delivery: At term, 12. Spontaneous, 9; midforceps, 2; low forceps, 1. Premature, 1. Spontaneous, eight months.

Weight at Birth: Heaviest, 3954 gm.; lightest, 2085 gm.; average, 3250 gm.

Length at Birth: Longest, 52 cm.; shortest, 46 cm.; average, 49 cm.

Malformations: Prealveolar Cleft, 5 cases. In 1 of these, a girl, the cleft was complete; in 4 boys the clefts were incomplete. All were unilateral. The cleft was on the left side in 2 boys and 1 girl; the side was not given in 2 boys.

Alveolar Cleft Lip, 1 case. In a boy there was a bilateral complete cleft of the lip with notching of the alveolar process on the left side.

Alveolar Cleft of Lip and Palate, 4 cases. In 1, a girl, there was a bilateral complete cleft of the lip, alveolar process and palate. In 3, all boys, there were unilateral clefts, one right and two left, of the lip, alveolar process and palate. Two of these were complete and 1 was incomplete.

Prealveolar and Postalveolar Cleft, 1 case. In this case, a boy, there was a unilateral left incomplete cleft of the lip with complete cleft of the hard and soft palate, the alveolar process being intact.

Postalveolar Cleft, 2 cases. In both cases, a boy and a girl, there were complete clefts of the soft palate, the alveolar process and lip being intact.

Associated Anomalies: 2 cases. In 1 boy there was a stricture of the ureter and hydronephrosis. (Died on fifth day.) In 1 girl there were bilateral club-feet and polydactylism of the right hand. (Died on first day.)

Mortality. 6 cases. One girl died on the first day, one hour after birth (bilateral club-feet, polydactylism). One boy died on the fifth day (stricture of the ureter and hydronephrosis). One boy died on the twenty-first day (bronchopneumonia, post-operative). One boy died on the twenty-fourth day (bronchopneumonia and inanition). One boy died on the thirty-fifth day (inanition). One girl died when eight months old (cause not given). In none of these cases was syphilis suspected or demonstrated.

SERIES C. WHITE CASES FROM THE OBSTETRICAL DEPARTMENT OF THE HOSPITAL FOR THE WOMEN OF MARYLAND

Number of Congenital Clefts of Lip and Palate in 3927 deliveries, 4.

Nationality of Mothers: United States, 4.

Ages of Mothers: Youngest, 27; oldest, 28, average; $27\frac{1}{2}$ years.

Health of Mothers: Good, 3; nasal sinusitis off and on during pregnancy, 1.

Mentality of Mothers: High grade.

Social Status of Mothers: High grade.

Ages of Fathers: Youngest, 26; oldest, 55; average, $37\frac{3}{4}$. In 1 instance the father was twenty-eight years older than the mother and in another fifteen years older. In 1 instance the mother was one year older than the father and in 1 instance the ages of the parents were equal.

Health of Fathers: Good, 4.

Social and Mental Status of Fathers: High grade, 4.

Primiparæ: 4.

Sex of Child: Male, 4.

Year of Birth: 1918, 2; 1919, 1; 1922, 1.

Legitimate: 4.

Presentation: L. O. A., 4.

Delivery. At term, 4. Spontaneous, 3; low forceps, 1.

Weight at Birth: Highest, 3615 gm.; lowest, 3153 gm.; average, 3322 gm.

Length at Birth: Not noted.

Malformations. Alveolar Cleft Lip and Palate, 3 cases. In 2 boys there were bilateral complete clefts of lip, alveolar process and palate. In 1 boy there was a unilateral left, complete cleft of lip, alveolar process and palate.

Postalveolar Cleft, 1 case. In 1 boy there was a cleft of the posterior third of the hard palate and all of the soft palate, the lip and alveolar process being intact.

Associated Anomalies, 1. In this case, a boy, there was unilateral club-foot on the right side (cleft on left side) and slightly bifid nose.

Mortality, 0. All of these cases were operated on and lived. The youngest is now two years old. Syphilis was not suspected or demonstrated in any of these cases.

General Summary of the Entire Series: *Number of Congenital Clefts of the Lip and Palate* in 28,085 deliveries, 24.

Nationality of Mothers: Nineteen were natives of the United States; 1 was a Bohemian; 1 was Irish; 1 was Roumanian and 2 were Russians.

Color. White, 17; black, 7.

Ages of Mothers: Second decade, 2; 17 and 19 years. Third decade, 15; 1 each, 22, 23, 25 and 28 years; 2 each, 20, 21, 24 and 26 years; 3 each, 27 years. Fourth decade, 7; 1 each, 33, 35, 36 and 38 years; 3 each, 30 years. The average age of the mothers of Series A was 26 years; in Series B was 25 years; in Series C was $27\frac{1}{2}$ years, with a general average of $26\frac{1}{3}$ years.

Health of Mothers: In the negro series it is probable, from the appearance of the child and placenta, that four mothers were syphilitic, although all tests were negative. All of these reported themselves in excellent health. One of the white mothers complained of "poor health" at the time of conception and another of tuberculosis of the lungs. Two mothers, one white and one colored, reported excessive vomiting during the first two and a half months of pregnancy. Otherwise the health of the mothers was excellent.

Mentality of Mothers: High grade, 4; ward type, 19; feeble-minded, 1.

Health of Fathers: This was noted in only 3 cases in the Johns Hopkins Hospital series, and all of these were in good health. In the 4 cases in Series C the health of the fathers was excellent.

Mentality of Fathers: High grade, 4; ward type in those mentioned, 3.

Primiparæ, 13; multiparæ, 11.

Sex of Child: White, male, 14; female, 3. Negro, male, 3; female, 4. Total, males, 17; females, 7.

Years of Birth: 1899, 1 case; 1902, 1 case; 1905, 2 cases; 1906, 1 case; 1907, 2 cases; 1909, 1 case; 1911, 2 cases; 1915, 3 cases; 1916, 1 case; 1918, 2 cases; 1919, 3 cases; 1920, 1 case; 1921, 2 cases; 1922, 2 cases.

Legitimate: White, 12; negro, 6. Total, 18.

Illegitimate: White, 5; negro, 1. Total, 6.

Presentation: L. O. A., 14; L. O. T., 1; R. O. A., 4; R. O. P., 4; not given, 1.

Delivery: In 21, delivery was at term; in 3, the birth was premature. The labor was spontaneous in 19 cases—13 white and 6 colored. Midforceps and low forceps were used in 2 cases each, all being whites. There was 1 version in a negro woman with a contracted pelvis. Of the 3 premature births, 1 was white, eight months, and 2 were colored, seven and a half and eight and a half months.

Weight at Birth: White: highest, 3954 gm.; lowest, 2085 gm.; average, 3263 gm. Colored: highest, 3317 gm.; lowest, 2268 gm.; average, 2648 gm.

Length at Birth: White: longest, 52 cm.; shortest, 46 cm.; average, 49 cm. Colored: longest, 53 cm.; shortest, 42 cm.; average, 47.7 cm.

Malformations. Prealveolar Cleft (cleft of the lip alone): The lip alone was cleft in 5 cases, all being in white children and all unilateral. One was complete (the cleft extending into the nostril) and 4 were incomplete. In 3, 2 males and 1 female, the

clefts were on the left side, and in 2 male children the side was not noted.

Alveolar Cleft Lip (cleft of the lip with notching of the alveolar process), 3 cases. In 1 negro girl the cleft was unilateral, complete with cleft and notch on the right side. In one negro boy the cleft was bilateral, complete with notching of both sides. In 1 white boy the cleft was bilateral, complete with notching of the alveolar process on the left side.

Alveolar Cleft Lip and Palate (cleft of the lip with cleft of the alveolar process, hard and soft palate), 11 cases. Seven were bilateral complete clefts—4 of these being colored, 2 boys and 2 girls; 3 white, 1 girl and 2 boys. Three were unilateral complete clefts, all being in white male children, 1 right and 2 left. One was a left unilateral incomplete cleft of the lip with complete cleft of the alveolar process and palate in a white boy.

Prealveolar and Postalveolar Cleft (cleft of the lip and palate, the alveolar process being intact), 1 case. In 1 white boy there was an incomplete left-sided cleft of the lip with complete cleft of the hard and soft palate.

Alveolar Cleft Palate (cleft of the alveolar process and palate, the lip being intact), 1 case. There was cleft of the alveolar process and palate in one negro girl. The side was not given.

Postalveolar Cleft (cleft of the palate alone), 3 cases. There was cleft of the posterior third of the hard and the soft palate in 1 boy and of the soft palate in 2 cases, 1 boy and 1 girl, all being white.

Associated Anomalies. In 6 cases there were other congenital anomalies, associated with lip and palate clefts. Three of these were in white children. One, a boy, had a stricture of the ureter and hydronephrosis and died on the twenty-fourth day; 1 girl, with bilateral club-feet and polydactylism of the right hand, died on the first day; 1 boy, who had a slightly bifid nose and a club-foot on the right side (the cleft being on the left side) still survives. Three were in negro children; 1 of these, a girl, had clubbing of the feet and hands and polydactylism of feet and hands, died on the fortieth day; 1 girl had enlarged thymus and

malformation of the mandible, was stillborn at term; 1 boy, a seven and a half months' macerated fetus, had a rudimentary hand and forearm and ankylosed elbow.

Mortality. Within eight months, 11 died out of the 24 cases. One was a macerated seven and a half months' negro male fetus; 1 was an eight and a half months' negro girl, who died after a few gasps; 1 negro girl was stillborn at term; 1 white girl died the first day, one hour after birth; 1 negro boy died the third day, luetic; 1 white boy died the fifth day, stricture of the ureter and hydronephrosis; 1 white boy died the twenty-first day, post-operative bronchopneumonia; 1 white boy died the twenty-fourth day, inanition and bronchopneumonia; 1 white boy died the thirty-fifth day, inanition; 1 negro girl died the forty-second day, inanition; one white girl died when eight months old, cause not given.

Comments. Five out of 24 of the mothers were of foreign birth, but this appears to have no significance. There is nothing noteworthy in the ages of the mothers. Bad health of the mother or father, or both, at time of conception and of the mother during the early months of pregnancy has been considered a possible cause for these malformations, as has also been excessive vomiting during the first two and one-half months of pregnancy, and these facts should be borne in mind. There was excessive vomiting during the first and second months in 3 instances. One mother had pulmonary tuberculosis at the time of conception. Another had "poor health" at the time of conception.

It has been claimed that syphilis is the definite factor in causing these malformations, and there was probably syphilis in 4 cases, all of these being in Series A. From this number (4 out of 7 cases) of apparently syphilitic children with congenital clefts of the lip and palate it might be inferred that syphilis played a definite part in the etiology, at least in the negro group. We must bear in mind, however, that the majority of syphilitic children, both black and white, are born without this malformation. In Series B and C no case of syphilis is recorded, and this seems to refute the idea, at any rate, for the white group.

Low-grade mentality of the parents has also been suggested as a possible cause of congenital clefts of the lip and palate. In the cases studied only one mother is listed as feeble-minded. The mentality of the others was sufficiently normal to excite no comment.

The difference in the ages of the parents has also been considered a possible etiological factor. In 1 instance the mother (feeble-minded) was twenty years old and the father (the girl's own father) was fifty-three years old, a difference of thirty-three years. In one instance the father was fifty-five and the mother twenty-seven; and in one instance there was fifteen years' difference. These were the most marked instances of difference in the ages of the parents in this series.

Maternal impressions were not recorded in the majority of the histories, and although interesting, they are of no particular importance as etiological factors.

Although no notes were found in the family histories of the occurrence of similar congenital clefts, heredity undoubtedly plays an important part in the occurrence of these clefts of the lip and palate. The percentage given by different authors varies between 15 per cent and 20 per cent. In my own cases I have noted about 19 per cent, with a family history of congenital clefts.

It has been said that these congenital malformations occur more frequently in illegitimate than in legitimate children. In this series, 18, or 75 per cent, were legitimate and 6, or 25 per cent, were illegitimate; all of these were in Series A and B. Inasmuch as one-fourth of the children were illegitimate, notice must be taken of this fact; but I question the importance of illegitimacy as an etiological factor.

Doubt has been expressed by those who seldom see negro patients as to whether congenital clefts of the lip and palate ever occur in this race. From case reports elsewhere and from the number reported in Series A we can conclude that the occurrence is not infrequent, but that it is not so common as in the white race.

The question of social status and environment is of considerable interest. The majority of the patients in the Johns Hopkins Hospital, Series A and B, were of the public ward and out-patient service type. Those from the Hospital for the Women of Maryland, Series C, were of the private ward class. It has been generally accepted that a greater proportionate number of these malformations occur among the children of individuals of the lower and more ignorant classes, whose nutrition and hygienic surroundings are poor, than among those whose environment is all that could be desired and whose mental attainments are of higher degree.

In 12,520 deliveries of negro women in Series A, 7 congenital clefts of the lip and palate were found, or 1 in 1788+.

In 11,638 deliveries of white women in Series B, 13 congenital clefts were found, or 1 in 895+.

In 3927 deliveries of white women in Series C, 4 congenital clefts were found, or 1 in 981+.

Taking the white series together, we find in 15,565 deliveries 17 clefts, or 1 in 915+; combining all the series in 28,085 deliveries there were 24 clefts, or 1 in 1170.

In other words, in Series A, where all conditions were most unfavorable, congenital clefts of the lip and palate occurred comparatively much less often than in Series B and C. It may be that these clefts occur less frequently in the negro race than in the white, irrespective of environment, etc.; but if this consideration is left out, these figures seem to upset the theory as to social status and environment. A comparison of Series B and C show that clefts occur in the white public ward cases (1–895+) more frequently than in the private ward cases (1–981+), but the comparative difference is not great when we take into consideration the vast contrast between the environment and social status of these groups.

We find that 13 out of 24, or 54+ per cent, of the entire series were first children. Eleven white mothers were primiparæ as compared with two negro mothers, which shows that the proportion of congenital malformations of the lip and palate were

greater in white primiparæ than in negro primiparæ. On the other hand, there was a greater proportion of negro multiparæ, 5 out of 7, as compared with the white multiparæ, 6 out of 17.

Haug, in 1904, collected from the literature 2352 cases of congenital clefts operated on in various surgical clinics and found that 64.3 per cent were males and 35.7 per cent were females. In this series there were 7 females, or 29.17 per cent, and 17 males, or 70.83 per cent. In other words, these clefts occur much more frequently in male than in female children.

The first delivery in the out-patient obstetrical service of the Johns Hopkins Hospital was on January 1, 1895, and the first congenital cleft occurring on this service was on July 14, 1899, or four and a half years later. The first delivery in the obstetrical ward of the Johns Hopkins Hospital was on August 17, 1896, and the first congenital cleft on this serivce occurred on July 22, 1902, or about six years later.

Nothing abnormal was noted during pregnancy in the type of presentation and in the course of labor in this series. Delivery was at term in 87+ per cent of the cases.

The average weight of a full-term normal white infant at birth is 3250 gm. The average weight in this series is 3263 gm., which is approximately normal. The average weight of a full-term normal negro infant at birth is 3104.8 gm. The average weight of this series is 2648 gm., which is considerably less than normal.

The average length of a full-term white normal infant at birth is 49.64 cm. The average length of this series of infants is 49 cm., which is normal. The average length of a full-term normal negro infant is 48.75 cm. The average length of this series is 47.7 cm., which is slightly shorter than normal.

From these figures it can be said that the average weight and length of a full-term white child with a congenital cleft of the lip and palate in this series of cases is equal to that of the normal white child at birth. In only 1 of the negro cases did the birth weight reach normal for this race. This may be accounted for by the state of health of the mothers, 4 out of 7 being probably syphilitic. Less favorable hygienic surroundings and possible

malnutrition during pregnancy may also have had some effect on the weight of the children of the negro series.

The lip was cleft in 20 cases, 83+ per cent of the series. In 5 of these, 25 per cent, the lip alone was cleft. In 15, or 75 per cent, there were in addition clefts of either the alveolar process or palate, or both. In other words, clefts of the lip complicated with clefts of the bony structure were found three times as often as simple clefts.

The palate alone was cleft in 3 cases, 12.5 per cent, and the alveolar process and palate in 1 case, 4+ per cent of the series. Therefore, clefts of the palate alone occurred less often (12.5 per cent) than simple lip clefts (20.8 per cent).

A comparison of the most severe types of clefts shows the following:

Alveolar cleft lip and palate (cleft of the lip, alveolar process and palate) occurred .4 times in 7 cases in Series A; 4 times in 13 cases in Series B and 3 times in 4 cases in Series C. Of those in Series A, all were bilateral complete, in Series B, 1 was bilateral and in Series C, 2 were bilateral.

It is noteworthy that in Series A, 57 per cent were in the bilateral group; in Series C, 50 per cent were in this group, while in Series B, only 7+ per cent were bilateral. Thus we find that the negro series showed the highest percentage of the bilateral type of cleft with the private ward, Series C, a close second.

Of the unilateral type in this same group there were 3 cases in Series B and 1 in Series C, and if we consider these in connection with the bilateral group, we find in Series A, 57 per cent; Series B, 30+ per cent; and in Series C, 75 per cent. In other words, in the series with the highest mentality and most favorable surroundings the comparative number of severe cases was greatest.

In the 20 cases with cleft of the lip, 2, or 10 per cent, were on the right side; 8, or 40 per cent, were on the left; 8, or 40 per cent, were bilateral and in 2, or 10 per cent, the side was not stated. This would make the occurrence of the clefts on the left side four times as frequent as on the right, and in this series the

bilateral clefts occurred in the same proportion, which seems unusually large.

The record of 11 deaths out of 24 cases (11 out of 20, or 55 per cent in Series A and B) is interesting, as it shows the mortality to be extremely high, even in the hospital where every facility is at hand for proper handling and care. It stands to reason that outside where facilities are frequently lacking the mortality must be at least as high, and is very probably higher. Syphilis, inanition and bronchopneumonia have been the principal causes of death.

A follow-up of the cases in Series B showed that one each was living fifteen, thirteen, three and two and a half years after leaving the hospital. In Series C, one each was living six, five and a half, five and two years after discharge. In Series A, none could be located.

It is interesting to compare the frequency of occurrence of congenital clefts of the lip and palate with other congenital malformations, and this was possible in Series A and B.

In 24,158 deliveries in the Johns Hopkins obstetrical service the following congenital defects, in addition to the 20 cases of cleft of the lip and palate, were found: Amnion adhesions, glaucoma, muscle defect, absence of esophagus, situs transversus and diaphragmatic hernia, 1 each; intrauterine amputation, cystic kidney, anencephalus, 2 each, or 1 in 12,079. Skeleton defect, teratoma, 3 each, or 1 in 8052+. Anus imperforate, hemicephalus, tracheoesophageal fistula, 4 each, or 1 in 6039+. Hernia, except umbilical, defects of intestines, 6 each, or 1 in 4026+. Defects of digits, defects of the external ear, 8 each, or 1 in 3019+. Webbed fingers, 9, or 1 in 2684. Hypospadias, nevus, 11 each, or 1 in 2196. Acrania, 14, or 1 in 1725+. Tongue-tie, 19, or 1 in 1271+. Hydrocephalus, 20, or 1 in 1207+. Spina bifida, 21, or 1 in 1150, and club-foot, 26, or 1 in 929+. Multiple digits, 134, or 1 in 180+. Umbilical hernia, 390, or 1 in 61+.

The above makes a total of 713, *i. e.*, one congenital defect (other than cleft lip and palate) in every 33+ children delivered.

This seems to be an extremely high percentage of defects, although the greater number were not of serious nature. We find that only spina bifida, club-foot, multiple digits and umbilical hernia occurred more frequently than congenital clefts of the lip and palate.

Conclusions. The incidence of congenital clefts of the lip and palate cannot be determined from a study of the admissions of these cases to surgical clinics or to institutions. Likewise, accurate data cannot be obtained on this point from the examination of male adults of draft age.

Definite conclusions cannot be drawn as to the relative importance of the various possible etiological factors, although in the negro series syphilis must be considered. Nothing unusual was noted during the course of pregnancy, labor and delivery, and the presentations were normal.

In 28,085 deliveries, 24 clefts of the lip and palate were found.

Congenital clefts of the lip and palate occur in the negro race, (in this series, 7 in 12,520 deliveries), but with less frequency than in the white (in this series 17 in 15,565 deliveries).

Environment and social status are apparently of little importance, as in the negro series, where conditions were most unfavorable, clefts occurred much less frequently (1–1788+) than in either white series. However, in the public ward white series clefts occurred more frequently (1–895+) than in the private ward series (1–981+). The incidence in all the series together was 1 in 1170+; in the two white series 1 in 915. These figures show a frequency of occurrence much greater than that estimated by Froebelius.

More than half the clefts were in first children.

The percentage of males was 70+, of females, 29+ per cent.

Clefts of the lip on the left side and bilateral clefts were found equally often, each occurring four times as often as right-sided clefts.

The lip was cleft in 83+ per cent of the cases. Of these in 25 per cent the lip alone was cleft; in 75 per cent there were, in addition, clefts of either the alveolar process or palate, or both.

The palate alone was cleft in 12.5 per cent of the cases and the alveolar process and palate in 4+ per cent.

In Series C, with the highest mentality and with the most favorable surroundings, the relative number of alveolar cleft lips and palates, the most severe type of cleft, was greater than in the other white series, 75 per cent as against 30+ per cent. This group of clefts occurred in 57 per cent of the negro series.

The average weight and length of a full-term white child with congenital cleft of the lip and palate are equal to that of the normal child at birth. The average weight of the negro child with congenital cleft of the lip and palate is considerably less than that of the normal negro child at birth, and the average length is slightly less.

Associated anomalies occurred in 25 per cent of the cases. This is a much larger percentage than we ordinarily find in cases admitted for operation. The mortality during the first few months is extremely high, 11, or 45+ per cent of the entire series. Syphilis, inanition and bronchopneumonia were the principal causes of death, although in addition 5 of these cases had associated anomalies.

Children with congenital clefts who live past the first year apparently have a reasonably good chance of surviving.

The occurrence of congenital defects other than those of the lip and palate, in the Johns Hopkins cases, is astonishingly high, one in every 33+ deliveries.

I realize that the number of cases studied in this series is too small to allow accurate conclusions to be drawn. However, certain information has been gained which is of considerable interest and value and which sheds light on the incidence of congenital clefts of the lip and palate, at least in this locality.

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DISCUSSION

Dr. Vilray P. Blair, St. Louis, Mo.:

I wish to thank Dr. Davis for his statistics. We have often wondered what relationship syphilis, for one thing, and prenatal environment had to do with clefts. His statistics on the subject are very interesting. In the average negro the cleft is very rare, but it does occur. In the great proportion of those in whom it did occur the child showed other evidences of malnutrition. The shortness of the child seems to me a strong argument that prenatal influence might have something to do with it, and we all know that inheritance has a lot to do with the condition. That one point was strongly brought out, and is of value.

A REPORT OF EIGHTY-SEVEN PRIMARY OPERABLE CASES OF CARCINOMA OF THE BREAST

ADMITTED TO THE NEW YORK HOSPITAL PRIOR TO APRIL 1, 1919

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AND

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The literature of mammary cancer contains many reports of high percentages of good end-results in the surgical treatment of this disease. A careful scrutiny of these records sometimes reveals the fact that many recent cases are included, making a proper estimate of the operative results difficult and at times impossible. The object of this paper is to place on record the late results in a series of cases of carcinoma of the breast operated at the New York Hospital[1] prior to April 1, 1919, five years or more ago. All cases of more recent date are excluded from consideration. A still more dependable period for end-results is seven or ten years, but sufficient cases are not available to make a report upon this basis. We appreciate that the group is a small one, but we feel that a critical analysis of these cases may furnish some valuable conclusions.

At the very outset we wish to place ourselves on record as objecting to the continued use of the term "three-year cure,"

[1] The writers gratefully acknowledge the privilege accorded them by Dr. Charles L. Gibson and Dr. Eugene H. Pool, of recording the cases of mammary cancer from the first and second surgical divisions of the hospital. They also appreciate similar courtesies extended by other surgeons on these services.

the basis of our objection being twofold: First, any serious consideration of results in the treatment of breast cancer at the end of three years is ill-advised, for the three-year limit gives a poor impression of what the end-result will be. Second, we desire to protest against the continued use of the term "cured" in recording the results of the treatment of this disease by any means. We are all familiar with the occasional cases of late recurrence, occurring many years after the surgical treatment of carcinoma of the breast, and therefore we believe that the word "cured" should be abandoned and replaced by the phrase, "No evidence of disease to date."

Our study embraces only primary cases, which were presumably operable and were subjected to surgery, in the hope of eradicating the disease. In general, the criteria of operability were a tumor in the breast, not fixed to the chest wall, with or without involved axillary nodes. The presence of supraclavicular nodes placed the case in the inoperable group, and no patient was subjected to a supraclavicular dissection. This judgment, concerning the involvement of supraclavicular nodes, is concurred in by Peck and Sutton, and Rodman and Greenough have stated that no satisfactory end-result has been attained where supraclavicular nodes were involved at the time of operation. We are so convinced that supraclavicular metastasis is an expression of a considerable dissemination of the disease, that today we call cases inoperable that show definite and well-marked supraclavicular fulness, even though no palpable nodes are present. Experience has taught us that such cases invariably develop nodes which are palpable a few weeks or months later.

The criteria of operability of mammary carcinoma are changing, and cases today are subjected to a much more careful scrutiny than was the case ten years ago. Therefore, it is probable that some of the patients included in this report and treated as primary operable carcinomas of the breast might today be placed in the inoperable class. The writers feel that the presence of axillary nodes, which are obviously extensively involved, reaching well up to the clavicle, or which are fixed to the chest wall, indicates inoperability.

Further, a more searching study of a case, with special regard to the paths and symptoms of distant metastases, especially to the chest and bones, will sometimes reveal evidence eliminating the patient from the operable class. A summary of the factors placing a case in the inoperable group has been indicated in a recent communication of one of the writers. We have not included in this study any of the primary inoperable or recurrent cases admitted to the hospital in the period mentioned, as we believe they must be considered separately. Further, the end-results in these groups have no relation to the problem of the treatment of primary operable mammary cancer to surgery.

If one weaves his way through the mass of literature upon this subject, he will find a varied series of figures of so-called "cures" in percentages varying from 22 per cent to 46 per cent. As one studies these statistics, he is forced to the conclusion that many factors must be considered in evaluating the reported results. These factors are:

1. The pathological diagnosis of carcinoma may vary widely in different clinics. If one includes in his carcinoma series borderline cases of epithelial growth (so-called precancerous lesions) the figures for good end-results will be raised to a very high percentage. As far back as 1907, Halsted pointed out that some pathologists might include "a few epithelial cells here and there escaping into the stroma as carcinoma." Are the cases of so-called "microscopical cancer," which Rodman says he finds in 23 per cent of the cases of chronic cystic mastitis, to be included in reports of end-results of the treatment of mammary cancer? We feel that the surgeon is too prone to accept as truth typewritten pathological reports made by pathologists, some of whom may lack the highest technical training. One of the writers has received pathological reports from one New York hospital upon five breast cases showing definite recurrent phases of mammary cancer, the report in all five instances being incorrectly rendered "fibroadenoma."

2. Many writers apparently include a certain proportion of recent cases, which swells percentage figures. We have found it

difficult to separate out the results in recent cases from those operated upon five years or more ago.

3. Many of the cases have not been completely followed up. Until the last decade, adequate follow-up systems were not a part of any hospital organization, and vast numbers of cases operated upon for this disease were completely lost track of. Therefore, it was quite impossible for most surgeons to evaluate the real end-results in the surgical treatment of cancer of the breast.

4. In many reports a large number of cases are excluded which have been lost sight of, many of whom are probably dead.

The present paper is a study of 87 presumably primary operable cases, 75 of whom have been followed through sufficiently to furnish accurate data concerning the five-year results obtained. Of the 75 cases the following tabulation is made:

RESULTS TO DATE IN 75 CASES OF PRIMARY OPERABLE CARCINOMA OF THE BREAST

Alive and with no evidence of recurrence	10
Died without recurrence more than five years after operation	1
Died with recurrence	54
Recurrence, but not completely traced	10
	75

A detailed list of the 75 patients appear on pages 280, 281 and 282.

Twelve cases are excluded from major consideration because of inconclusive data as to end-results, but these patients are separately studied in conjunction with the 75 complete cases in tabulating certain etiological data. These 12 patients group themselves into two classes: namely, those dying before five years had elasped without recurrence, but from intercurrent disease, and, secondly, those which were impossible to trace, although not recurrent when last observed. A brief tabulation of these 12 cases is appended on page 283.

A study of the 87 cases yields certain facts concerning the

influence of trauma, previous abscess in the breast and prior lactation in connection with the etiology of mammary cancer.

Trauma. Of the entire 87 cases a positive or negative statement concerning a definite history of trauma was made by 55 patients. In each instance a positive statement by the patient was considered reasonably reliable evidence.

TRAUMA AS AN ETIOLOGICAL FACTOR

Positive.		Negative.	
No.	Per cent.	No.	Per cent.
15	27	40	73

The traumatizing agent varied from blows or falls upon the breast to corset pressure, and one patient made the statement that she had been accustomed for years to stick pins into the portion of the breast which subsequently became the seat of cancerous disease.

TYPES OF TRAUMA

Blow or fall upon breast	11
Corset pressure	2
Bullet wound	1
Habit of sticking pins in breast	1
	15

Previous Abscess of the Breast. Five cases had previously suffered from abscess of the breast. There seems little question that the damage done to mammary tissue in the presence of a suppurative process furnishes favorable soil for the development of carcinoma.

Prior Lactation. We are more and more impressed with the frequency of occurrence of cancer in breasts that have never lactated. In this series no statement concerning previous lactation was made in 15 instances. Of the remaining 72 patients, exactly one-half had a history of previous lactation. It seems reasonable to conclude that prior lactation is not an important factor in the development of mammary cancer.

PRIOR LACTATION

Positive.	Per cent.	Negative.	Per cent.
36	50	36	50

No.	Surgeon.	Year.	Name.	Age.	Duration.		Total duration.	Result.	Pathology.	Date of metastasis.	Metastasis.
					Before operation.	After operation.					
1	Hitzrot	1912	S.	45	2 yrs.	2½ yrs.	4 yrs. 6 mos.	Died	Scirrhus	6 mos.	Opp. breast, spine.
2	Lee	1913	C.	26	5 mos.	11 mos.	1 yr. 4 mos.	Died	Duct carcinoma	4 mos.	Opp. breast, chest, supraclavicular, axilla.
3	Lee	1913	H.	50	6 mos.	3 mos.	9 mos.	Died	Scirrhus	3 mos.	Spine.
4	Gibson	1913	L.	34	8 mos.	5 mos.	1 yr. 1 mo.	Died	Carcinoma very malignant	2 mos.	?
5	Gibson	1913	M.	52	7 mos.	2 yrs. 8 mos.	3 yrs. 3 mos.	Died	Scirrhus	?	?
6	Gibson	1913	V.	58	1 yr.	10 yrs. 5 mos.	11 yrs. 5 mos.	A. W.	Scirrhus		
7	Lee	1913	C.	49	1 yr 6 ms	9 mos.	2 yrs. 3 mos.	Died	Scirrhus	2 mos.	Chest, supraclavicular.
8	Farr	1913	K.	49	....	....	.....	A. W.	Carcinoma		
9	Gibson	1913	L.	41	....	....	.....	Died	Medullary carcinoma	14 mos.	Sternum.
10	Lee	1914	K.	45	1 yr. 6 mos.	5 yrs. 9 mos.	7 yrs. 3 mos.	Died	Scirrhus	Without	recurrence
11	Lee	1914	M.	53	5 mos.	1 yr. 8 mos.	2 yrs. 1 mo.	Died	Scirrhus	1 yr. 6 mos.	Chest wall.
12	Lee	1914	H.	48	5 yrs.	2 yrs. 2 mos.	7 yrs. 2 mos.	Died	Scirrhus	2 yrs.	Spine.
13	Gibson	1914	C.	57	....	....	.....	A. W.	Scirrhus		
14	Gibson	1914	S.	50	6 mos.	1 yr. 6 mos.	2 yrs.	Died	Carcinoma	1 yr. 3 mos.	Upper axillas cupraclavicular nodes.
15	Hitzrot	1914	N.	47	2 yrs.	5 yrs. 4 mos.	7 yrs. 5 mos.	Died	Carcinoma	4 yrs. 6 mos.	Right supraclavicular, chest.
16	Lee	1915	M.	46	1 yr.	1 yr. 1 mo.	2 yrs. 1 mo.	Died.	Duct carcinoma	3 mos.	Cervical nodes.
17	Gibson	1915	O.	46	5 yrs.	9 yrs.	14 yrs.	A. W.	Carcinoma		
18	Lee	1915	G.	60	8 mos.	4 yrs. 5 mos.	5 yrs. 1 mo.	Died	Carcinoma	?	Supraclavicular nodes.
19	Hitzrot	1915	S.	51	2 yrs.	11 mos.	2 yrs. 11 mos.	Died	Simplex	4 mos.	Scalp.
20	Farr	1915	A.	47	8 mos.	1 yr.	1 yr. 8 mos.	Died	Medullary carcinoma	10 mos.	Chest wall.
21	Lee	1915	Y.	45	6 mos.	5 yrs. 6 mos.	6 yrs.	Died	Carcinoma	5 yrs.	Supraclavicular. Lumbar spine.
22	Lee	1915	O.	43	1 yr.	5 yrs. 7 mos.	6 yrs. 7 mos.	Died	Carcinoma	5 yrs.	Spine.
23	Lee	1915	F.	46	8 mos.	5 yrs. 10 mos.	6 yrs. 6 mos.	Died	Large alveolar	2 yrs.	Spine.
24	Turune	1915	C.	38	7 mos.	18 mos.	2 yrs. 3 mos.	....	.....	6 mos.	Chest wall incomplete.
25	Bancroft	1915	S.	28	9 mos.	1 yr.	1 yr. 9 mos.	Died	Scirrhus	7 mos.	Supraclavicular.
26	Bancroft	1915	S.	41	9 yrs.	7 yrs. 6 mos.	16 yrs. 1 mo.	Died	Scirrhus	22 mos.	Supraclavicular.

27	Pool	1915	P.	51	4 yrs.	6 mos.	4 yrs. 6 mos.	Died	Carcinoma	4 mos.	Supraclavicular.
28	Lee	1916	E.	39	6 mos.	7 yrs. 5 mos.	7 yrs. 11 mos.	A. W.	Adenocarcinoma		
29	Lee	1916	C.	40	1 yr.	2 yrs. 9 mos.	3 yrs. 9 mos.	Died	Scirrhus	9 mos.	Same breast.
30	Pool	1916	S.	53	4 yrs.	1 yr. 6 mos.	5 yrs. 6 mos.	Died	Carcinoma	Few mos.	Supraclavicular nodes, later general.
31	Hitzrot	1916	G.	40	11 mos.	6 yrs.	6 yrs. 11 mos.	Died	Carcinoma	3 yrs.	Skin, chest.
32	Bancroft	1916	B.	41	1 yr. 6 mos.	1 mo.	?	Recurrent	Carcinoma	1 mo.	Supraclavicular nodes.
33	Erdman	1916	Y.	60	4 mos.	6 mos.	10 mos.	Died	Fibro-adeno-carcinoma	3 mos.	Supraclavicular nodes.
34	Bancroft	1916	G.	16	4 yrs.	8 yrs.	12 yrs.	A. W.	Fibro-adeno-carcinoma		
35	Erdman	1916	P.	47	6 mos.	8 mos.	1 yr. 2 mos.	Died	Carcinoma	?	Axilla, general metastasis.
36	Pool	1916	P.	67	1 yr.	3 yrs. 6 mos.	4 yrs. 6 mos.	Died	Scirrhus	....	Lung metastasis.
37	Gibson	1916	F	63	1 wk.	5 yrs. 1 mo.	5 yrs. 1 mo.	Died	Carcinoma	3 yrs. 1 mo.	Opposite breast and axilla, amputation.
38	Lee	1916	N.	49	2 mos.	7 yrs. 4 mos.	7 yrs. 6 mos.	Died	Carcinoma	6 mos.	
39	Lee	1916	M.	54	2 wks.	1 yr. 5 mos.	1 yr. 5 mos.	Died	Carcinoma	6 mos.	Scar. Later gen. metastasis.
40	Gibson	1916	J.	60	2 yrs.	?	?	Recurrent	Scirrhus Carcinoma	5 yrs. 7 mos.	Axilla (could not complete).
41	Gibson	1916	F.	55	2 wks.	2 yrs. 1 mo.	2 yrs. 1 mo.	Died	Scirrhus carcinoma	2 yrs.	Spine.
42	Gibson	1916	D.	78	6 mos.	4 yrs. 3 mos.	4 yrs. 9 mos.	Died	Scirrhus carcinoma	4 yrs.	Gen. carcinoma.
43	Farr	1916	R.	36	9 mos.	1 yr. 7 mos.	2 yrs. 4 mos.	Died	Carcinoma	1 yr. 5 mos.	Same breast, neck, chest.
44	Hitzrot	1916	S.	53	10 days	2 yrs. 6 mos.	2 yrs. 6 mos.	Died	Carcinoma	1 yr. 11 mos.	Chest, abdomen.
45	Farr	1916	S.	35	10 days	7 yrs.	7 yrs.	A. W.	Scirrhus carcinoma		
46	Hitzrot	1916	M.	39	1 yr.	1 yr. 2 mos.	2 yrs. 2 mos.	Died	Scirrhus carcinoma	7 mos.	Bones.
47	Farr	1917	W.	28	2 mos.	1 yr. 1 mo.	?	Recurrent	Carcinoma	1 yr. 1 mo.	Line excision, left axilla.
48	Gibson	1917	B.	60	6 mos.	2 yrs. 5 mos.	2 yrs. 6 mos.	Died			
49	Bancroft	1917	S.	40	4 mos.	3 yrs. 7 mos.	3 yrs. 11 mos.	Recurrent	Gelatinous	3 yrs. 7 mos.	5–6 costal cartilages.
50	Gibson	1917	H.	52	1 wk.	4 yrs. 9 mos.	4 yrs. 9 mos.	Died	Carcinoma	4 yrs.	Brain, tibia.
51	Lee	1917	R.	35	1 yr.	10 mos.	1 yr. 10 mos.	Died.	Carcinoma	6 mos.	Axilla, breast and axilla.
52	Farr	1917	M.	40	10 mos.	11 mos.	1 yr. 9 mos.	Died	Carcinoma	8 mos.	Local.

No.	Surgeon.	Year.	Name.	Age.	Duration		Total duration.	Result.	Pathology.	Date of metastasis.	Metastasis.
					Before operation	After operation.					
53	Hitzrot	1917	B.	56	2 mos.	1 yr.	1 yr. 2 mos.	Recurrent	Scirrhus carcinoma	9 mos.	Uterus, omentum.
54	Gibson	1917	M.	62	3 yrs.	4 mos.	3 yrs. 4 mos.	Died	Scirrhus carcinoma	?	?
55	Lee	1917	M.	63	2 wks.	3 yrs. 4 mos.	3 yrs. 4 mos.	Died	Scirrhus carcinoma	3 yrs. 4 mos.	Abdomen.
56	Lee	1917	K.	70	9 mos.	1 yr. 4 mos.	2 yrs. 1 mo.	Died	Scirrhus carcinoma	8 mos.	
57	Farr	1917	L.	37	5 mos.	5 mos.	10 mos.	Died	Scirrhus carcinoma	2 mos.	Abdomen.
58	Gibson	1917	C.	43	?	4 yrs. 3 mos.	?	Died	Carcinoma	?	Spine, axilla.
59	Bancroft	1917	D.	30	3½ mos.	3 yrs.	3 yrs. 3½ mos.	Died	Fibrocarcinoma	2 yrs. 8 mos.	Spine.
60	....	1917	L.	45	5 wks.	2 yrs.	2 yrs. 1 mo.	Recurrent	Carcinoma	1 yr.	Sternum.
61	Farr	1918	B.	43	1 yr. 6 mos.	3 yrs. 2 mos.	4 yrs. 8 mos.	Died	Scirrhus carcinoma	3 yrs.	Brain, lung.
62	Gibson	1918	O.	54	3 mos.	2 yrs. 9 mos.	3 yrs.	Died	Fibrocarcinoma	2 yrs. 10 mos.	Same axilla.
63	Gibson	1918	H.	59	7 mos.	1 yr. 3 mos.	1 yr. 10 mos.	Died	Adenocarcinoma	8 mos.	Intra-abdominal.
64	Gibson	1918	H.	53	1 yr. 6 mos.	4 yrs. 8 mos.	6 yrs. 2 mos.	Recurrent	.....	4 yrs.	
65	Farr	1918	S.	36	1 mo.	6 mos.	7 mos.	Died	Adenocarcinoma	2 mos.	Right breast, lung, liver, spine.
66	Gibson	1918	M.	58	?	2 yrs. 6 mos.	?	Recurrent	Fibrocarcinoma	2 yrs. 6 mos.	Skin.
67	Hitzrot	1918	C.	45	1 yr.	3 yrs. 6 mos.	4 yrs. 6 mos.	Died	Fibrocarcinoma	2 yrs. 5 mos.	Skin, lungs, ribs.
68	Hitzrot	1918	N.	54	1½ yrs.	4 yrs. 4 mos.	5 yrs. 10 mos.	Died	Scirrhus carcinoma	4 yrs.	Chest.
69	Bancroft	1918	B.	60	3 wks.	5 yrs. 6 mos.	5 yrs. 7 mos.	A. W.	Scirrhus		
70	Erdman	1918	C.	43	7 mos.	5 yrs. 10 mos.	6 yrs. 5 mos.	Recurrent	Fibrocarcinoma	3 yrs. 6 mos.	Right axilla.
71	....	1918	H.	44	1 yr.	8 mos.	1 yr. 8 mos.	Died	Large alveolar	5 mos.	Liver.
72	Hawkes	1918	T.	41	1 yr.	8 mos.	1 yr. 8 mos.	Died	Small alveolar	Few mos.	General metastasis.
73	....	1918	S.	40	3–4 mos	5 yrs.	5 yrs. 4 mos.	A. W.	Carcinoma		
74	Hawkes	1919	F.	60	3 mos.	5 yrs. 1 mo.	5 yrs. 4 mos.	A. W.	Adenocarcinoma		
75	Gibson	1919	A.	68	2 mos.	2 yrs. 7 mos.	2 yrs. 9 mos.	Died	Medullary carcinoma	2 yrs. 10 mos.	Site operation.

DIED BEFORE FIVE YEARS POSTOPERATION WITHOUT RECURRENCE

No.	Surgeon.	Year.	Name.	Age.	Duration.		Total duration.	Result.	Pathology.	Date of metastasis.	Metastasis.
					Before operation.	After operation.					
76	Bancroft	1917	B.	38	8 days	1 mo. 4 days	2 mos.	Died	Carcinoma	1 mo.	Same side.
77	Bancroft	1918	L.	56	5 wks.	11 days	6 wks.	Died	Carcinoma	11th postoperative day. Lobar pneumonia.	
78	Erdman	1918	T.	46	2 mo.	1 yr. 6 mos.	1 yr. 8 mo.	Died	Fibrocarcinoma	Insane. Died on Ward's Island. No recurrence.	
79	Erdman	1918	U.	51	6 wks.	2½ wks.	8 wks	Died	Comedocarcinoma	Pneumonia 6 days after leaving hospital.	
80	Gibson	1919	W.	33	?	?	?	Died	Carcinoma	Insane shortly after discharge. No recurrence.	

INCONCLUSIVE (NOT FOLLOWED), NOT RECURRENT WHEN LAST SEEN

No.	Surgeon.	Year.	Name.	Age.	Duration.		Total duration.	Result.	Pathology.	
					Before operation	After operation.				
81	Gibson	1913	D.	44	1 mo.	1 yr. 7 mos.	?	?	Carcinoma	Lost track of.
82	Lee	1914	M.	45	1 yr.	2 yrs. 9 mos.	?	?	Carcinoma	Lost track of.
83	Lee	1914	M.	38	1 yr.	....	.....	.....	Adenocarcinoma	
84	Lee	1915	Z.	66	8 mo.	2 yr. 4 mos.	?	?	Carcinoma	Lost track of.
85	Lee	1916	F.	40	1 yr.	....	?	?	Papillary cyst	
86	Hitzot	1917	C.	50	1 yr. 6 mos.	1 yr. 3 mos.	?	?	Carcinoma	Lost track of.
87	Gibson	1918	M.	42	....	2 yrs. 2 mos.	.....	.....	Fibrocarcinoma	Lost track of.

Situation of the Tumor. In 81 cases the record shows that the left breast was involved in 43 instances and the right breast in 38.

A description of the portion of the breast involved is given in 69 instances. The following table indicates the location of the tumor:

Upper outer quadrant	33
Lower outer quadrant	12
Outer half	8
Mesial above nipple	8
Upper inner quadrant	4
Lower inner quadrant	3
Upper half	1
Total	69

Correct Preoperative Diagnosis of Carcinoma of the Breast. A record of the preoperative diagnosis was recorded in all instances. Percentage of correct diagnosis is shown in the table below.

CORRECT PREOPERATIVE DIAGNOSIS OF CARCINOMA OF THE BREAST

No. of cases correct.	Per cent.	No. of cases incorrect.	Per cent.
81	93	6	7

All surgeons appreciate the difficulty in making a correct diagnosis in every instance. The percentage indicated in the table above is practically identical with that reported by Mills[14] and Greenough[6] and Simmons,[6] the former giving 93 per cent and the latter 94 per cent.

Preoperative Diagnosis of Involved Axillary Nodes. In 4 instances no statement was made as to the preoperative impression of metastasis to the axilla. In 83 patients definite statements are recorded, giving the preoperative diagnosis of the surgeon as to the involvement of axillary nodes.

PREOPERATIVE DIAGNOSIS OF INVOLVED AXILLARY NODES

	No.	Correct.	Per cent, correct.
Believed to be positive	59	49	83
Believed to be negative	24	19	79
	83		

This tabulation illustrates the difficulty of correctly diagnosing involvement of axillary nodes. The experience is universal to occasionally encounter considerable axillary metastasis where none was anticipated, and *vice versa*, to find hyperplastic lymph nodes free from cancerous disease where definite malignancy was expected. Although deductions from a small group of cases are always dangerous, the table shows a lessened liability of error where the surgeon believes the nodes to be definitely involved.

Factors Influencing Prognosis. Various factors must be considered in reaching a correct prognosis in any case of cancer of the breast. Each patient represents a complex problem due to the variable conditions afforded by age, the rapidity of growth, associated pregnancy, the pathological type of tumor, the presence or absence of involved axillary nodes, the proper interpretation of the signs of early chest metastasis and of early metastasis to bone. We will briefly consider each of these factors:

Age. Of the 75 cases completely followed, we have divided them into three age groups, namely:

	No.	No. alive.	Per cent, alive.
I. Up to and including forty years of age . .	19	4	21
II. Forty-one to and including sixty years of age	49	7	14
III. Sixty-one years and over	7	0	0
	75		

I. *Patients up to and Including Forty Years of Age.* All authorities agree that, other things being equal, the younger the patient the more serious the outlook. In the present series the percentage in this age group alive and without disease five years or more after operation (21 per cent) is distinctly higher than the average for the entire group. The reason for this high percentage in the younger women can be explained, partially, at least, by the fact that all of the 4 living cases in this group were distinctly localized tumors without metastasis to the axilla. Further, Case 34 presented some very unusual pathological features, which will be discussed under the section on pathology, making it perhaps questionable whether this case should be included at all in the present study. If this case were excluded the percentage alive would be 16 per cent rather than 21 per

cent. The small number of cases available for study in this age group makes any percentage figure inconclusive.

II. *Patients from Forty-one to and Including Sixty Years of Age.* We feel that no special comment upon this group is necessary other than to call attention to the fact that it represents the approximate cross-section of percentage results five years after surgical treatment of the disease.

III. *Patients Sixty-one Years of Age and Over.* Experience has generally proven that patients in this group, as a rule, do well following surgical intervention. Usually the rate of growth is slow and the patients are apt to live many years following operation before menacing metastases occur. The group of 7 cases is much too small from which to draw any conclusive deductions, but it perhaps illustrates that this group may not be as favorable as we have generally believed it to be.

Rapidity of Growth. In general, a convincing statement by the patient that the growth of the tumor has been exceedingly rapid should lead the surgeon away from rather than toward surgical intervention. We have frequently heard surgeons express the opinion that immediate radical operation should be undertaken, because the growth has been at such a startling rate. Such an attitude we believe to be an incorrect one, as the prognosis of these rapid cases, treated by radical surgery, is always bad, and we believe many of them would survive the disease longer if some other form of therapy were followed. If the growth is rapid and the woman is below forty, in general we feel that operation is unwise. Practically without exception, the patients with rapidly growing tumors in the present series have succumbed to the disease within a few months up to two years of the date of operation.

Associated Pregnancy. We are all thoroughly familiar with the extreme gravity of the prognosis in a case of carcinoma of the breast associated with pregnancy. These patients are usually young women, which adds further to the seriousness of the prognosis. In our series one patient (Case 2), aged twenty-six years, was seven months pregnant at the time of her admission to the hospital. Radical operation was advised and done by one of the

writers with a rapidly disastrous result, the patient surviving the surgical intervention by but eleven months. The result in this case represents a judgment against the overenthusiasm of youth. Today no such management of the situation would for a moment be entertained. The almost immediate recurrence with the rapid extension of the disease was frightful. This patient finally died in the hospital with a huge massive involvement of the whole chest wall, extensive adjacent metastasis to the lung and pleura and ended with an opening several inches across, entering the chest cavity.

The presence of a lactating breast associated with carcinoma is far less menacing to the patient than pregnancy. Three patients in this report were nursing children at the time of the breast amputation. Case 24 developed a recurrence in six months and died eighteen months postoperative, with extensive chest metastasis. Cases 59 and 70 also recurred, the first two years and eight months and the latter three years and six months after operation. The pathological report in each instance was a fibrocarcinoma, which probably accounts partially for the delay in recurrence. Further, Case 70 received considerable prophylactic *x*-ray treatment over a period of two and a half years, which probably helped to delay the date of recurrence and the rate of growth of the tumor process.

Pathological Types. Sistrunk and MacCarty and many others have pointed out the wide variations in pathological types of mammary cancer as to the degree of malignancy. We are convinced that the term "carcinoma of the breast" in reality covers a group of diseases presenting widely different clinical courses and frequently distinctive pathological pictures. We believe that as more accurate knowledge of these pathological groups is obtained the results of the treatment of cancer of the breast can be much better standardized. At the present time we are convinced that many cases are placed in the carcinoma group which really do not belong there. It is often difficult to properly interpret a pathological picture even when the interpretation is made by pathologists of the front rank. Case 34, in this present series, illustrates the difficulty in question. This

young woman, aged sixteen years, was operated upon for what was believed to be a fibroadenoma. A local removal of the tumor was practised, and upon the basis of the pathological examination by Elser, corroborated by Ewing, a local mastectomy was performed. A diagnosis of fibro-adeno-carcinoma was rendered, but in the experience of both of these pathologists the tumor is quite unique. Sections of the tumor show nodules composed of a cellular epithelial growth which in part is of glandular type. These nodules are sharply defined by an unbroken limiting membrane. The surrounding fibrous tissue shows considerable overgrowth, with some disorder in arrangement. More recently, in reviewing this slide, both Elser and Ewing expressed grave doubt whether or not one should include such a case in a group of cases of mammary carcinoma. If a more systematic effort were made to gather together unusual cases of this and other types, we feel that it might ultimately be possible to separate out cases of this sort from the cancer group.

The following table is appended, setting forth the number of cases of each pathological group encountered in this series. Although a pathological report was rendered in each instance, in 27 cases the diagnosis of "carcinoma" only was made. The percentages of each pathological type in the 48 remaining cases is given. The percentage alive for each group is also added in a separate column:

PATHOLOGICAL TYPES

	No.	Per cent.	No. alive five years without recurrence.	Per cent of group alive.
Scirrhus carcinoma	26	54	5	19
Fibrocarcinoma	5	10	0	0
Adenocarcinoma	4	8	2	5
Alveolar carcinoma	4	8	0	0
Medullary carcinoma	3	6	0	0
Fibro-adenocarcinoma	2	4	1	50
Duct carcinoma	2	44	0	0
Carcinoma simplex	1	2	0	0
Gelatinous carcinoma	1	2	0	0
	48	98[1]	8	

[1] The 2 per cent missing is accounted for by our eliminating from consideration any fractional percentages to facilitate interpretation of the table.

Presence or Absence of Involved Axillary Nodes. Of the 75 conclusive cases, pathological examination of the axillary contents proved the axillary nodes to be free from disease in 30 patients. Ten of these cases, or exactly 33 per cent, are alive and well at the present writing. This percentage is very much lower than the usual figure given by most writers, as will be seen from the following table:

PERCENTAGE OF GOOD RESULTS IN CASES WITHOUT AXILLARY INVOLVEMENT

Surgeon.	Per cent alive without recurrence.
Lockwood	83 (4 years or more)
MacCarty and Sistrunk	65 (5 to 8 years)
Mills	63 (6 years)
Greenough and Simmons	56 (5 years)

It is a startling fact that only one patient of the entire 75 cases survived the five-year period without metastasis, where axillary nodes were found involved by the pathologist. This patient, Case 10, died without evidence of recurrence, of an intercurrent disease five years and nine months after operation. The almost uniformly bad result in the entire group following radical amputation in the presence of involved axillary nodes is the most striking feature developed by our study of this series.

Proper Interpretation of Signs of Early Chest Metastasis. We believe that few surgeons today appreciate the early physical and radiographic signs of chest metastasis. Craver has pointed out that physical signs of early chest metastasis consist in "a peculiar limitation of breath sounds, especially marked during inspiration, covering a limited area of the chest. This may or may not be accompanied by fine, crackling rales during inspiration or by pleural friction rubs." It is only in the later stages of chest involvement that such subjective symptoms as shortness of breath and a dry, hacking cough may be expected. The radiographic appearance of early chest metastasis may be characteristic a considerable time before the appearance of any subjective symptoms. We feel certain that chest plates are not

infrequently passed as negative when true evidence of extension of the disease to the chest is revealed in the *x*-ray plate. Surgeons, as well as radiologists, often look for pronounced shadows, which are seldom seen early in the course of chest metastasis. In a joint communication with Herendeen, one of the writers has pointed out that the evidence of early metastasis consists "of hazy, line-like streaks along the bronchi, extending in a radiating manner from the hilum out into the parenchyma of the lung. It is usually bilateral and is generally more pronounced upon the side corresponding to the primary tumor. One may also see ill-defined mottling which in more marked cases gives almost the appearance of miliary tuberculosis. There may also be noted enlargement of the nodes at the hilum of the lungs."

Symptoms of Metastases to Bones. Pain is the first symptom of metastasis to bones, and the patient may complain before the *x*-ray plate will reveal definite evidence of disease. The surgeon should be alert in investigating thoroughly any complaint of pain by the patient, especially in the region of the ribs, spine, pelvic bones or femur. Vague rheumatic symptoms should arouse the suspicions of the surgeon, and a careful physical examination may reveal metastasis to bones in a patient who might otherwise be considered operable. In the order of frequency bones involved are: spine, pelvis, ribs (usually multiple), femur and other bones.

In the present series metastasis to the spine was known to have occurred in eight patients.

General Paths of Metastasis. As most of the patients succumbing to the disease died outside of the hospital, no satisfactory statement can be made concerning the situation or extent of metastasis. A considerable experience with mammary carcinoma has demonstrated that the most frequent path of metastasis is into the axilla of the same side, extending upward, and ultimately involving the corresponding supraclavicular nodes. Metastasis into the chest is common and sometimes occurs early. Metastasis to the spine is unfortunately common and makes its appearance in the natural course of the disease, independent of any

surgical intervention. The vast majority of cases coming to autopsy show almost invariably intrathoracic metastasis and often involvement of the liver.

Site of First Recurrence. In 60 patients a note is made of the position of the first recurrence following operation. The subjoined table gives the percentage figures for the recurrent areas:

	Per cent.
Supraclavicular (same side)	21
Chest	19
Spine	11
Axilla (same side)	10
Adjacent skin	10
Intra-abdominal metastasis	9
Bones other than the spine	8
Opposite breast	6
Opposite axilla	3
Brain	2
Distant skin	1
	100

These figures can only give an approximate idea of the first situation in which evidence of the disease may be expected following operation. The careful follow-up of the present day would reveal a very much higher percentage of metastasis to the chest.

Treatment. As surgeons we have been accustomed to focus rather more upon the type of operation performed than upon the disease we are called upon to treat. Warren, in 1904, called attention to the fact that the result of the surgical treatment of mammary cancer depended largely upon the degree of malignancy of the process. Since the time when the modern radical operation devised independently by Halsted[9] and Willy Meyer came into vogue, surgical treatment of this disease has been carried out with a high degree of technical skill. We have realized what a serious prospect faced the patient unless a complete removal of all malignant neoplastic tissue could be accomplished. Still, in spite of the excellent surgery performed in the treatment

of carcinoma of the breast, the problem of its complete control remains most difficult.

The astonishingly good results which are frequently obtained by some form of incomplete operation have impressed many operators. Greenough and Simmons obtained 26 per cent of good five-year results, while in our own series 50 per cent of the cases treated by local mastectomy have remained well without evidence of disease up to the present time. Mills reports 9 out of 14 cases, or 64 per cent, well and without disease six years postoperative. If we consider for a moment the reason for this splendid showing for the incomplete operation, it will be immediately apparent that the cases upon which this type of operation has been performed are usually those of a relatively low grade of malignancy, or cases in which the diagnosis of carcinoma before operation was questionable. Pathologists generally appreciate that papillary cyst adenocarcinoma, although grouped with the malignant neoplasms of the breast, is a relatively benign disease, and local mastectomy or even a wide local removal of the tumor may give a satisfactory end-result in such a case.

In future surgeons should and will use the radical operation as the routine procedure for the majority of cases, but in exceptional instances a somewhat less extensive type of surgery may yield equally good results.

In this series but 10 of the patients had received postoperative *x*-radiation, and most of these cases were treated by a technic which today is considered only reasonably efficient. Nevertheless, the average duration of life of these patients was three years and four months after operation. None of the 10 were favorable cases, as axillary nodes were involved in each instance. The average length of life of 61 patients who received no radiation was three years, and this group included all of the favorable cases without metastasis to the axillary nodes. These figures are suggestive but not conclusive.

In the cases under consideration there was one operative mortality, Case 77, dying on the eleventh postoperative day,

from lobar pneumonia. The following table gives the operative mortality statistics in the hands of various operators:

OPERATIVE MORTALITY

Surgeon.	Mortality, per cent.	
Leech	7.0	
Greenough and Simmons	3.6	1894–1904
Halsted	3.6	
Warren	2.0	
Buchanan	1.5	
Lee and Cornell	1.0	
Sistrunk and MacCarty	0.5	
Crile	0.0	
Lockwood	0.0	
Greenough and Simmons	0.0	1911–1914

Late Results. The following table illustrates the marked variation in the statistics of good end-results furnished by different surgeons.

Surgeon.	No. of cases.	Per cent alive five years without recurrence.	
Haggard and Douglass	111	46	
Sistrunk and MacCarty	218	39	
Peck and White	69	39	
Tixier	150	38	(6 years)
Bunts	248	33	
Greenough and Simmons	95	32	
Lindenberg	183	28	
Wiesman	106	23	
Ochsner	98	22	
Lee and Cornell	75	15	

The graphic chart on page 294 illustrates the rapid fall of percentages alive and well at six-month intervals down to the five-year period in the cases in this report:

Conclusions. 1. Five-year results in the treatment of mammary cancer are the shortest ones worthy of report.

2. The five-year results in this series show 15 per cent alive and with no evidence of disease.

3. The term "cured" should be abolished and supplanted by "no evidence of disease to date."

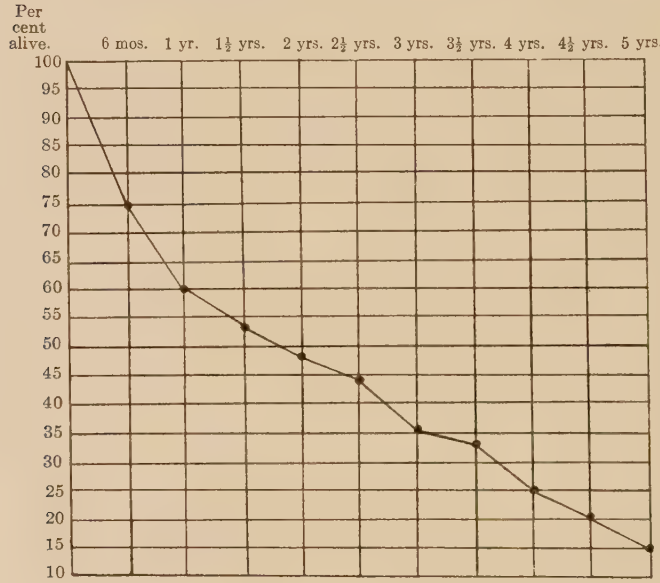
4. Criteria of operability must be more sharply drawn.

5. The surgeon must learn to appreciate the physical and radiographic signs of early chest metastasis.

6. Metastasis to bones must be carefully excluded in every presumably primary operable case.

7. Continual follow-up of all postoperative cases is necessary if correct figures are to be obtained.

TABLE SHOWING PER CENT ALIVE WITHOUT RECURRENCE AT SIX-MONTH INTERVALS UP TO FIVE YEARS



8. The term carcinoma of the breast probably includes a group of diseases differing in their pathology and their degree of malignancy.

9. Unusual pathological types should be collected in order that they may be separated out, the non-malignant from the truly cancerous.

10. In cases with involvement of axillary nodes, radical amputation usually yields disappointing end-results.

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DISCUSSION

Dr. Robert B. Greenough, Boston, Mass.:

I think Dr. Lee is to be congratulated on his very clear statement of the present situation in regard to cancer of the breast. Conditions have changed materially in the last ten years in regard to this disease. One reason for this, I believe, is the use of the *x*-rays and radium, particularly the *x*-rays, which have made it possible for the surgeon to be much more selective in his choice of cases for operation.

I should like to emphasize the fact that in my opinion bad axillary involvement is much better not operated on. A patient with extensive axillary metastasis will live longer and more comfortably under *x*-ray treatment than if a futile attempt is made to remove the disease by operation. I do believe in the removal of a sloughing mass of tumor on the chest wall, but beyond that I would abandon operative procedures in favor of the *x*-ray in advanced cases.

In regard to the use of the term "cure" in a case of cancer, there have been various ways in which people have attempted to get around the fact that no case of cancer can ever be said to be positively cured, and if we use the term with that reservation, I see no reason why we should not apply it just as well at the end of five years as at the end of seven, or at the end of ten years, for there may still be recurrences even after that period.

We have studied our cases by means of a chart such as Dr. Lee has shown, and we have found that the percentage of cases free from recurrence, which is, of course, 100 per cent at the time immediately following operation, drops very rapidly during the first three years to about 32 per cent, and in the cases of our series, which we followed for at least six and a half years, this percentage then flattened out and remained above 30 per cent to the end of the period. There will undoubtedly always be some cases of late recurrence, but the percentage of these in any series which has gone three or five years without recurrence will be relatively small.

In regard to the condition of the lung in cases of cancer of the breast, Dr. Lee has made a point which I think is not universally acknowledged or understood, namely, that the preoperative study of any case of cancer of the breast where operative treatment is under consideration should always include a very careful *x*-ray examination of that patient's lung. Changes in the lung which are not at all demonstrable by any other methods of physical examination can be detected by the *x*-ray, and for that reason I fully believe that every clinic should make it a routine to have a proper examination of the chest with the *x*-ray before the decision for or against operation is determined.

Dr. Crile, Cleveland, Ohio:

I should like to ask Dr. Lee what effect, if any, radiation has on the prognosis?

Dr. Willy Meyer, New York:

We all must feel grateful to Dr. Lee that he has followed up his cases so carefully. Fortunately, most of us are today in the same position at our hospitals. We recall how difficult it was in former years to follow up ward patients; it was almost entirely impossible with their ever-shifting domicile, so that for many years I followed up only my private patients. I have seen that since we have been carrying out the true radical operations, Nos. 1, 2 and 4 of the series operated on respectively in 1894, 1895 and 1896 are still alive and well today without any sign of recurrence. Every one of these patients had infiltrated axillary nodes. If I understood Dr. Lee's chart correctly, he does not consider these cases adapted to radical operation. I believe that in the first and second stage of cancer, that is, where there is a tumor of the breast and glands in the axilla, radical operation is absolutely indicated.

In speaking of supraclavicular nodes we must not forget that there are 3 or 4 cases on record where patients who had these nodes above the clavicle have remained well for quite a number of years. Halsted, Rodman, Perthes, etc., had each one or two such cases. To refuse operation to a patient who has nodes above the clavicle may be correct in many cases, but it means passing final judgment and telling the patient that there is no more hope in operation. I think this is rather severe. We should not try the effect of *x*-rays and radium in such cases and not refuse such a patient a primary operation. That at least has been my practice. The radical neck operation is not such a terrible thing. It is done in all operations for carcinoma of the neck, of the face and skull very frequently, and, therefore, I personally would not exclude these cases definitely from such interference.

Dr. C. H. Mayo, Rochester, Minn.:

It seems to me that Dr. Lee has drawn the line a little too close—in fact, has leaned backward in presenting the absolute truth. An analysis of what he has presented would mean that there is always a chance for mistaken diagnosis, as he has shown that the question of prognosis would depend largely on the pathological report. I believe it is a good thing to take such a report and have the pathologist go over it. The report is very discouraging for the physician as well as the patient. In cases of cancer of the breast, roentgenograms should

be taken of the whole chest. In this way certain groups of cases formerly accepted as operative risks would be eliminated and properly classified.

Dr. Ellsworth Eliot, Jr., New York:

I am very much in sympathy with Dr. Lee in regard to the question of "cures" after operation. While I quite agree with Dr. Mayo that some interval must be agreed upon, say five years, to serve as a criterion of operative success, I regard it as desirable that all such cases as survive at the end of that interval should be reported as "free from recurrence" rather than as operative "cures."

In the second place I believe that it is necessary to distinguish between actual recurrence and a fresh outbreak of the disease independent of the primary focus. In a paper read by me two years ago, before this Association, this particular phase of the subject was discussed. While as was stated yesterday, many cases of this character are observed in the colon, as, for example, where a primary growth of the splenic flexure was followed by a carcinoma of the sigmoid, and *vice versa*, the breast is no exception to such an unusual clinical history. In this connection I venture to report the further progress of a patient whose history was presented in the paper already referred to. A case of scirrhus carcinoma of the breast was followed eighteen months after operation by the development of a similar growth in the opposite gland. At neither operation was there any involvement of the axilla. The first operation was done fifteen, and the second thirteen and a half years ago. Recently, the patient was found to have developed consolidation of the right lower lobe without pleural effusion and without constitutional symptoms. Whether this is a reappearance of the carcinoma or not is quite doubtful. At least the limitation of this lesion to one lobe as demonstrated by the *x*-ray is most unusual.

In conclusion, I wish to call attention to the fact that, in New York, the treatment of carcinoma by the high-frequency current has been advocated by certain members of the medical profession even when the primary growth presents a most favorable condition for radical cure. I believe that such a procedure in all cases of operable malignancy, with its consequent delay of months, cannot be too strongly condemned, and I sincerely hope that this point of view may be further emphasized by other members of this Association. In one such instance of primary carcinoma of the breast no change in the character of the growth was noticeable after six months' treatment by this method. The patient was then referred for operation with the recommendation that only that part of the breast involved in the

growth should be sacrificed and that the axilla should not be disturbed as any involved glands had been eradicated by the high-frequency current. This advise was not followed, the usual complete operation being done, removing the entire breast and axillary contents. On subsequent microscopical examination it was found that the high-frequency application had destroyed in places the connective-tissue framework of the growth, permitting the extravasation of viable cancer cells into the contiguous tissue spaces. Furthermore, operation disclosed the presence of involved glands in the axilla which, on microscopical examination, were found to present the same changes as obtained in the primary growth.

Dr. A. C. Wood, Philadelphia:

I should like to add a few words to what Dr. Eliot has said, and to add my testimony to the importance of wise discrimination in the selection of these cases that are absolutely and indisputably operable.

A patient was referred to me two years ago, but as it happened I left the city the day before for my summer holiday, and the next move was to take the patient to one of the best-known radiologists of this country. The radiologist said, "I believe that I can cure this tumor by radiating with *x*-rays," and he gave such a positive assurance to the family that they accepted his advice. The patient was treated for some months, and finally the radiologist said: "I feel that this is not going to get well. You had better have an operation." I operated on the patient. I took the specimen to Dr. Allen J. Smith, the pathologist of the University of Pennsylvania, told him the history, said the patient had been radiated very extensively for between three and four months, and said "will you, in your histological examination, make a careful study of the tissue and see if you can find any effect at all that has been produced by the radiation; in other words—can you find anything at all in the histology of this tissue that is different from what you have been looking at for thirty years in a case of malignancy that has not had any radiation?" He reported that he could not see any difference at all. There was nothing in the specimen to indicate that any influence had been produced by the radiation. Certainly, the tumor had not been affected; the axillary glands were involved. The patient is still alive and without recurrence. She was operated on in November, 1922.

Dr. William B. Coley, New York:

I think Dr. Lee has given us a very valuable paper upon a most important subject. Dr. Mayo has made a very good suggestion as to

the importance of getting *x*-ray pictures of the chest before operating upon these patients with cancer of the breast. I do not think we can set aside the more optimistic statistics dealing with the end-results of operation for cancer of the breast by such men as Halsted, Willy Meyer, Primrose, Sampson-Handley, Rodman and Haggard, showing nearly 50 per cent of their series well beyond the three-year period following radical operation. Many years ago W. Mitchell Banks, in the *British Medical Journal*, reported 250 cases of cancer of the breast treated by operation, with special reference to recurrence. Only 26 per cent of the cases that had remained well for a period of three years developed a recurrence after that period; in other words, if a woman with cancer of the breast has remained well for three years after operation, there are three chances out of four that she will remain permanently well. In some instances, however, a local recurrence has developed a long time after the operation. Personally, I have observed one case in which there was a local recurrence seventeen years later. Sir Anthony Bowlby once told me that he had observed a metastatic carcinoma in the supraclavicular gland thirty-two years after the breast had been removed for carcinoma. These cases, however, are exceptions and should not play any part in determining the value of operative procedure, and we have practically every reason to regard as permanently cured any patient who has remained well for five years after the operation.

Dr. Eliot has brought up an important point, and that is the danger of using radium or *x*-rays before operation in operable cases. At present there is a growing tendency on the part of surgeons to employ preoperative radiation. Dr. Greenough, in his paper on "Cancer of the Breast" (read before the American Cancer Research Society, in Boston, 1923) emphasized the disadvantages of this procedure, the chief disadvantage being the delay in performing the operation for removal of the breast; and, second, the interference with the normal healing of the wound caused by the preliminary *x*-ray treatment. Furthermore, Dr. Greenough stated that he had not seen in his own experience, nor had he been able to find reported in the literature, a single case of carcinoma of the breast that had been cured by *x*-ray or radium.

Dr. Lee fortunately has a series of cases of operable carcinoma of the breast in which, for various reasons, such as the presence of intercurrent disease or unwillingness on the part of the patient to submit to operation, radium was used, and Dr. Lee will be able to tell us, in a very few years, whether or not he has been able to cure such patients without operation. At present I do not believe that either radium

or *x*-ray should be used in cases of operable carcinoma of the breast; on the other hand, a great many men are using *x*-ray for three or four weeks before the operation as a preoperative measure. We have no data, in my opinion, to justify us in doing this, and I think there is a strong possibility, if not a probability, that during this three- or four-weeks period metastases may develop which otherwise would not have been the case had an immediate operation been performed.

Dr. F. C. Wood, New York:

With regard to the application of the *x*-ray to these patients, I quite agree that there is no evidence at the present time of the possibility of curing cancer of the breast by *x*-ray. I hold very strongly to the opinion, with which, however, others do not agree, that preoperative radiation of the breast is a mistake, and my reasons for this belief is that, in the first place, we know that any dose which can be put through the skin and still leave that skin in a healthy condition will damage but a small portion of the cells of the tumor. In many instances it will not cause any perceptible shrinkage of the tumor. We also know that any radiation which can be applied to the axilla will not destroy carcinomatous nodes. If, on the other hand, a sufficient dose is given the skin is irreparably damaged and healing of the wound doubtful.

Another point which Dr. Coley has brought up, and with which I agree, is that every minute of delay after the diagnosis of cancer has been made is an additional danger to the patient.

We pathologists often see the extensive vascular involvement of the small capillaries in breast tumors, and the experimental work in my own laboratory has shown that it is very easy to massage particles into the circulation. After a single cell has reached the lung, all surgery becomes merely a palliative measure.

One important fact we do know is that the moment the diagnosis is made, the tumor, if it is operable, should be removed and any preoperative radiation is a pure delay which is disadvantageous to the patient and no advantage to the surgeon.

Dr. Miles F. Porter, Fort Wayne, Ind.:

I have had some experience recently which has led me to believe that the preoperative treatment by radiation tends to increase the necessity for drainage and delays the union of the wounds. I can say positively that it seemed to me that in those cases where any preoperative treatment by radiation was had the drainage has been longer continued, as if there had been a breaking down of the subcuticular tissues,

and in two or three cases there has been a delayed union of the wound. Whether that means an argument in favor of radiation and that the cancer cells are breaking down, I do not know, but I have certainly been impressed with it. Personally, I like to get along withour drainage in many of these cases of radical operation of cancer of the breast. I have failed to get as satisfactory results without drainage since I have been using the *x*-ray as a preliminary treatment, and I should like to know what the experience of others has been along the same line.

Dr. Lee (in closing):

I just want to emphasize one or two points. I feel that we are rather overoptimistic in our judgment of the results of treatment of this disease, and there are numerous experiences illustrating our unjustifiable optimism which I could recite to you had I the time.

In many hospitals these patients are run through so quickly, often entering late in the afternoon, with radical amputation the following morning, that a quiet, critical judgment may be impossible. I see them four or five weeks later, with marked supraclavicular nodes or definite evidence of chest metastasis.

I feel certain that frequently such cases are not well scrutinized. I believe that few radiologists and very few surgeons recognize the real picture of early chest metastasis. The appearance is characteristic, but the untrained eye will pass as normal many a case which is really inoperable.

Most of the discussion upon this paper has been relative to radiation. I wanted to present these cases from the purely surgical viewpoint, for none of them had received adequate postoperative radiation, and in no instance had preoperative radiation been given. It was a clean-cut surgical end-result which I desired to bring before you.

As to preoperative radiation, I do not think any one has any data of any sort for or against preoperative radiation. We have been using it at the Memorial Hospital for several years as a routine. We do not know whether it is or is not of value. Up to the present time no one can say that it is not of value, because no one has reliable data upon which to base a judgment. When we can compare in sufficient numbers the cases in which it has been used with those in which it has not been used, then we may be able to state with confidence whether it is or is not of value.

I want to emphasize once more that if the surgeon will go over his cases more carefully, he will probably find many instances of carcinoma of the breast which have already passed beyond the operable stage, although the first impression seemed to indicate a favorable setting for surgery.

THE ADDITIONAL POSTERIOR INCISION IN CERTAIN CASES OF OPERATION FOR THE INFLAMED RETROCECAL APPENDIX

By WILLY MEYER, M.D.
NEW YORK

There can be no question that it is to the advantage of the patient under operation if the surgeon can see plainly what he is doing. Starting from this premise, I have made it a rule, in my personal work, even as far back as the early times of the evolution of the appendix chapter, thirty years ago, to expose to view the inflamed organ in its entire length in the course of its extirpation. I never practised the method of working through a small incision and trying to separate adhesions bluntly by the mere sense of touch when operating for appendicitis.

I have applied this rule also to the so-called retrocecal appendix when the latter is tied down, often by old and firm bands, to the posterior surface of the cecum and ascending colon, and cannot be brought into view by any kind of manipulation.

In this type of cases I have found it expedient, and to the best interest of patient as well as surgeon, to abandon the anterior incision, perrectal or pararectal, and add a posterior one which exposed the retrocecal and retrocolonic space, without entering the retroperitoneal tissue.

If McBurney's intermuscular operation had been in progress, then the additional posterior incision was made a part of the former.

Every surgeon has met with instances in which the appendix could not be brought forward and properly exposed for the usual tying of the mesoappendix and consecutive steps. He then follows downward to the cecum the presenting longitudinal band

of the ascending colon. But not infrequently just the very base of the appendix alone appears; its further course, for the moment, is not discernible. Then on lifting the caput coli with the left hand and slipping the right forefinger around the cecum we often feel the mass behind the gut. In many cases, when the organ lies entirely within the peritoneal cavity[1] (Fig. 1), we succeed in pushing it forward and bringing its tip into view,

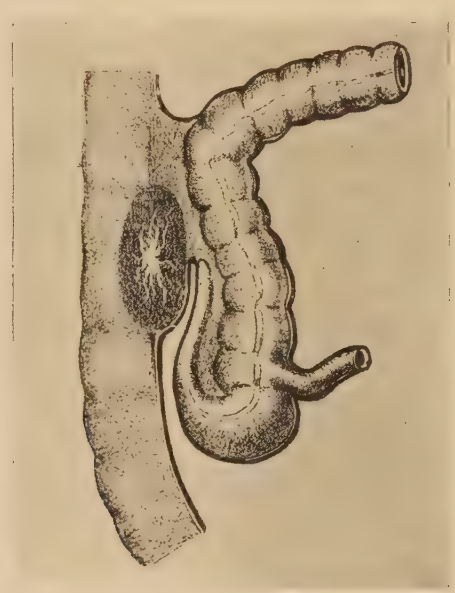

FIG. 1.—Reproduced from G. S. Huntington, l. c. Tip of retrocecal appendix reaches level of right kidney, but is completely covered with peritoneum. In such an instance the surgeon usually is able to mobilize the inflamed organ and make its distal end appear alongside the ascending colon.

which then renders easy the completion of the operation. In other instances this maneuver is unsuccessful. Inflammatory bands of old standing are unyielding; firm adhesions or congenital anatomical conditions make the appendix absolutely immovable, it forming a unit with the intestine, or congenital anatomical relations hold it fixed posteriorly[1] (Figs. 2 and 3).

[1] Ileocecal folds and fossæ from, George S. Huntington, the Anatomy of the Human Peritoneum, Plate cclxxxi, p. 272.

In such an emergency, in acute and subacute as well as chronic cases, the surgeon has to decide quickly as to the best and safest procedure in the given case.

Personally, I believe it is to the patient's interest not to insist upon forcing the further steps of the operation by way of the original incision, but to place a sandbag under the patient's right

FIG. 2.—See G. S. Huntington, l. c. The greater portion of the retrocecal appendix, commencing at its base, runs upward toward the kidney within the retroperitoneal tissue, its distal third, however, is situated within the general peritoneal cavity, a fold of peritoneum, descending from above, covering it all around.

hip and proceed as follows: Map out the middle between McBurney's point and the anterior superior spine by placing the thumb and third finger on these two points and the forefinger upon the center of the imaginary line, connecting the umbilicus with the anterior superior spine, and from here make a second incision upward through the skin toward the region of the kidney, about two and a half to three inches long (Fig. 4). After

dividing the fascia of the external oblique muscle and its muscular substance in the same direction, the fibers of the internal oblique are cut across, bleeding vessels being immediately caught and tied. Transverse fascia with parietal peritoneum appears, in stout patients clearly marked by a layer of fat; they are also cut. Now the retrocecal and retrocolonic space has been opened and

FIG. 3.—Also copied from G. S. Huntington, l. c. The entire long retrocecal appendix is situated outside of the peritoneal sac; its tip again reaches up to about the middle of the kidney. Blunt manipulation from within the peritoneal cavity, without guidance of the eye, must necessarily represent a hazardous surgical procedure.

its condition can be explored under the guidance of the eyes. Usually I have made this wound of the abdominal parietes funnel-shaped, the deep incision shorter than that of the skin, in order to cut across as few fibers of the internal oblique as possible. I always open the peritoneal cavity in the lower angle of the wound first, because the entrance into the peritoneal sac at this place is assured. According to requirements the whole

FIG. 4.—Showing the two incisions as used by the author in cases of adherent retrocecal appendix which cannot be properly reached and loosened from the front. The one nearest the middle line represents the typical pararectal incision, which is abandoned in case of necessity, the second then being added; the lower end of the latter corresponds to the middle between McBurney's point and the anterior superior spine and then runs backward and upward as long as required, dividing the muscles sharply.

FIG. 5.—Typical McBurney incision which, in spite of being lengthened intermuscularly, does not give proper access in order to completely expose a long appendix which runs behind of and upward with cecum and ascending colon. The same incision is added as shown in Fig. 4 commencing at the middle of the upper border of the exposed and separated muscle substance. It will permit to do any work required under the surgeon's eyes, no matter how long, twisted and adherent the appendix.

incision can be lengthened upward in the course of the further work. With the borders of the wound well held apart by means of broad blunt retractors the appendix usually comes quickly into view and can now be followed up in its entire course to its tip and be safely and correctly dealt with.

If McBurney's intermuscular operation had been done the posterior incision starts from the middle of the superior lip of the transverse muscular and peritoneal wound and runs from there upward (Fig. 5), the inner and outer end of the original peritoneal incision having been marked by clamps, which are left temporarily in place.

Drainage and closure of the posterior wound is done according to general rules. If in acute or subacute cases pus was encountered, due to gangrene, with an infiltrated mesoappendix, the cigarette drain will be a safeguard, as in other cases of acute appendicitis, and the desired drainage is facilitated on account of the directly downward course of the canal, the patient being on his back.

In the absence of gangrene we close up completely by means of layer sutures. The anterior wound is always closed airtight.

I first mentioned this procedure, which I did not find described in the literature, when discussing Dr. A. S. Vosburgh's paper, "Non-rotation of the Intestine; its Relation to High Retrocecal and Aberrant Position of the Appendix," read before the New York Surgical Society on October 9, 1912.[1] Later on I elaborated on it in the "Festschrift" dedicated to Prof. Fr. Trendelenburg by his former assistants and his successor in the Chair of Surgery at the University of Leipsic on the occasion of his seventieth birthday.[2] On November 5, 1920, I read a paper on the same subject, entitled "A Safe and Simple Method for the Extirpation of the Adherent Retrocecal Appendix," before the Surgical Section of the New York Academy of Medicine. This has not been published so far.

[1] Annals of Surgery, December, 1912.

[2] Zur Chirurgie des Wurmfortsatzes, Deutsche Ztschr. f. Chir., 1914, cxxix, 321.

A recent instructive personal experience brought the value of the additional posterior incision, as above described, again forcibly to my mind, and impelled me to write this paper and discuss the method before our Association.

FIG. 6.—A high McBurney incision; the lower end of the wound corresponds to the omphalospinous line. It will permit to thoroughly expose to the inspection of the surgeon the appendix which is situated retrocecally, provided it does not run too far upward, and cecum with colon are movable. If need be, the upward sharp incision can be added as shown in Fig. 5.

I prefer not to cite this last case in spite of its many points of interest, because it was somewhat atypical and did not, owing to pathological peculiarities encountered, show all the advantages of the additional posterior advance, as I had seen them in previous experiences. I shall therefore pick out for illustration just a very few cases from a series of upward of 30 of this type that have been operated upon by me on the basis of the above-cited plan within the last twenty-eight years.

According to my personal records the additional posterior incision for an adherent retrocecal appendix was done by me for the first time in 1896. As a matter of evolution variations of the operation were developed according to the character of the inflammation—acute, subacute, chronic—as well as to the possibility of a correct diagnosis previous to operating regarding the position of the inflamed organ; that is to say, in a number of instances access was gained by means of the posterior incision primarily, sharp through all the muscles, or by the intermuscular approach, at the place and in the direction mentioned above for the additional posterior incision, or by a high intermuscular approach according to McBurney (Fig. 6), leaving out the preliminary entrance into the abdominal cavity from the front. The variations are:

1. Anterior incision with additional lumbar cut.

2. McBurney's intermuscular operation with additional sharp incision backward and upward from the middle of the upper lip of the musculoperitoneal wound.

3. Primary McBurney intermuscular incision, high above the omphalospinous line, without the necessity of an additional posterior incision.

4. Primary posterior sharp incision through all the three muscles constituting the abdominal wall.

The two latter varieties will not be discussed here.

I. Pararectal or perrectal incision plus additional lumbar incision.

Male, aged forty-eight years. In May, 1904, sudden severe colic, rectospasm, vomiting. Admitted to Lenox Hill Hospital ten hours after the onset of the attack.

Operation. Pararectal incision; appendix not found; incision lengthened upward; cecum high; insertion of appendix exposed, runs in retrocecal direction; extensive adhesions; discontinuance of operation by this route; temporary tamponade; right side of pelvis raised upon a sandbag; sharp incision backward through the three muscle layers (as above described), which had gradually to be lengthened to the anterior tip of the twelfth rib; with

a second cushion placed under the lumbar spine, producing pronounced lordosis, the entire very long appendix comes finally into view; from its insertion into the cecum it turns backward and upward toward the renal region; thence toward the middle line, and from there downward again close to its starting point, thus describing about nine-tenths of a circle. Ligation of old, strong adhesions step by step; appendectomy; suture of wound; cure.

Female, aged thirty-five years; perforative gangrenous appendicitis.

Great sensitiveness over McBurney's point, extending upward toward the border of the liver. Admitted to Lenox Hill Hospital October 27, 1905. Pararectal incision; peritoneal cavity filled with pus; appendix does not come into view on pulling upon the cecum. It is therefore assumed that the organ is located retrocecally. Additional posterior incision with sharp division of muscles, as in the preceding case. Appendix seen at once; very long; firm adhesions to the posterior surface of the ascending colon; tip near the liver found to be gangrenous and perforated; suture with drainage; cure.

Male, aged twenty-seven years; acute gangrenous appendicitis.

Seriously ill for twenty-four hours; tenderness posteriorly and in front; some rectospasm. Admitted to Lenox Hill Hospital May 20, 1919. Immediate operation: Pararectal incision; only base of gangrenous appendix visible; organ retrocecal and firmly adherent; additional posterior incision exposes a totally gangrenous appendix; removed without breaking the organ; anterior and posterior cut closed; rubber tissue drain in upper end of lumbar wound; uninterrupted recovery.

II. McBurney's blunt intermuscular incision with additional sharp incision backward and upward from the middle of the upper lip of the muscular wound.

Boy, aged twelve years; interval operation at Lenox Hill

Hospital May 14, 1901. McBurney's blunt, intermuscular division; cecum not found even after placing the patient in Trendelenburg's posture; intermuscular incision continued inwardly (Weir); wide access; sigmoid flexure to the right; cecum away up, tightly adherent; appendix runs posteriorly and upward, equally firmly adherent; impossible to reach for good surgical work, therefore posterior incision through muscles added; organ now fully accessible, thickened, firmly adherent to the intestine and omentum; loosening; excision; suture; cure.

Male, aged twenty-five years; acute appendicitis.

Two weeks previously peculiar attack of intra-abdominal inflammation: Diagnosis of appendicitis doubted by attending physician on account of the alleged absence of tenderness over McBurney's point. Moderate sensitiveness posteriorly at time of consultation. Diagnosis: Appendicitis; organ situated posteriorly. Operation December 12, 1906, at Lenox Hill Hospital. Blunt division of muscles (McBurney) above the anterior superior spine; the appendix does not come into view; the finger palpates organ tightly adherent, posteriorly; on attempting to loosen it, pus appears; incision lengthened inwardly (Weir), good access, but appendix cannot be reached as yet; closure of inner two-thirds of wound; sharp division of muscles posteriorly and upward, with sandbag under right half of pelvis. It becomes evident that a perityphlitic abscess had advanced posteriorly into the lumbar muscles, the suppurating canal being about two inches long; appendix itself very short, funnel-shaped at insertion near cecum and contains thin feces; excision; drainage; recovery.

Of course, the surgeon can try in cases of this type to do away with the sharp division of the posterior abdominal wall in one and the same line and proceed by means of a high placed McBurney's intermuscular operation with Weir's addition, for a wide exposure is usually required. However, cases will be encountered as I have just described them, where the cecum is situated so high that even a *very* wide intermuscular approach will still be found insufficient, and the sharp division of the

muscles upward will become imperative. And it may be done without hesitancy, because neither important nerves nor blood-vessels are cut. Personally, I have not observed a subsequent abdominal hernia in a single instance, even when, in former years, wide drainage with gauze tampons was done. The careful suture of the wound in layers with the insertion of a cigarette drain or a twisted piece of rubber dam, as practised nowadays, makes the development of an abdominal rupture well-nigh impossible. In one of the cases the local tonus of the abdominal wall was found to be impaired, evidently on account of the division of branches of the twelfth intercostal or the ileohypogastric nerve which cross the field; here the right hypogastric region was somewhat bulging for a while, but a hernia did not develop. Such sequelæ, of course, can sometimes not be avoided and must be faced when difficult life-saving work is to be safely carried out.

In emergencies such as described, some surgeons practise primary division of the base of the appendix, as mentioned above, and, after proper attendance to its stump, follow the course of the organ toward its tip. I have tried this myself, but cannot see any advantage in such a course. It certainly is the better plan to try to work up from the base to the tip with the organ remaining attached to the cecum, and after complete exposure make the division of the base of the appendix the last step of the operation. In particularly difficult cases this procedure will also not lead to a successful removal of the entire appendix except the parietal peritoneum alongside the large intestine is incised.

Others abhor the idea of making a second abdominal incision in an operation for appendicitis, no matter how complicated the case may prove to be. They cut through the fold of the parietal peritoneum parallel with and on the outer side of the cecum and ascending colon. This allows the operator to pull the large intestine inward and turn its posterior side up. However, it seems to me that this procedure is far less advantageous for the patient, surely less advantageous in case of acute gan-

grenous inflammation of the rectrocecal appendix, with perforation and pus formation. Such an incision along the outer side of the gut usually enters the retroperitoneal space and is then bound to open new avenues for absorption and possible additional infection at a time when Nature, in many instances to be sure, had just succeeded in closing them, due to the intense inflammation present. Besides—and this is the principal drawback—such incision intentionally unites the general peritoneal cavity, which in some cases, on account of the anatomical conditions present, so far was but slightly or not at all involved,[1] with the region of the source of inflammation. Whereas by adding the second posterior incision we reach the focus of intensest disease by the shortest and most direct route. We are able to loosen and remove the appendix, no matter how long and twisted it may prove to be, with ease and precision, watching every step, without coming in contact with the general peritoneal cavity, and we can drain, should this appear necessary, in a most direct and natural way.

Proceeding as above described, the surgeon will often be surprised to observe how the removal of a firmly adherent inflamed retrocecal appendix, which at first glance appeared to be a difficult and complicated task technically, with one stroke becomes transformed into a comparatively easy and safe operation.

[1] Confer G. S. Huntington, loc. cit.

AN EXPERIMENTAL CONSIDERATION OF THE INFECTION OF PERMANENT HYDRONEPHROSES

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AND
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The underlying cause in the development of a hydronephrosis is generally recognized to be any factor that causes obstruction to urinary outflow from the kidney or ureter. This obstruction may be complete and constant, in which event a permanent hydronephrosis develops, or it may be transient or incomplete, when an intermittent or moderate hydronephrosis is present. In either event, the obstruction to free urinary outflow is one of the chief factors in causation of infection of the urinary stream, it being necessary only to add the offending organism to produce the picture of a urinary-tract infection of mild or severe type. In view of the many and various causes of hydronephrosis, varying from an aberrant renal vessel or a congenital ureteral valve to absolute obstruction of the ureter, it is the purpose of this paper to study some of the factors which may be of importance in converting a relatively harmless hydronephrosis into a pyonephrosis.

In order to have a fixed condition to study, we have elected to consider infection of hydronephroses due to complete obstruction of the ureter rather than the intermittent or incomplete types, though the principles governing the infection of all types are more or less the same. Clinically, the permanent hydronephroses are usually due to accidental or purposeful ligation

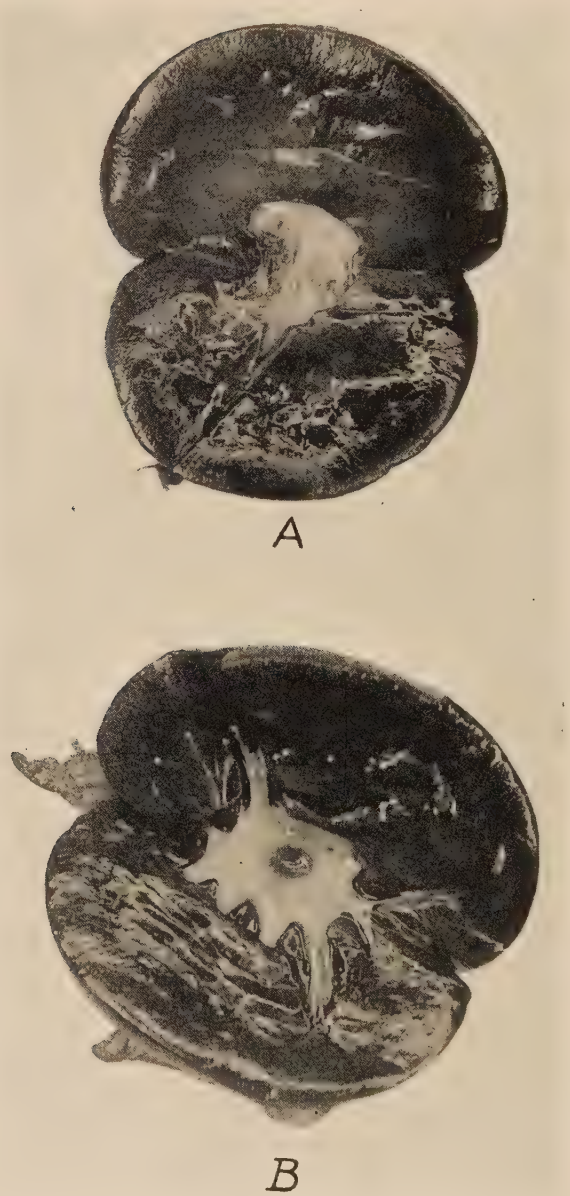

FIG. 1.—Dog 7-7. Twelve-hour hydronephrosis after ligation of ureter. *A*. Normal kidney. *B*. Beginning hydronephrosis.

of a ureter during an operative procedure in the pelvis, pressure on the ureter from inflammatory masses or any growths, stricture of the ureter, obstruction by stone, products of inflammation or congenital anomalies.

Barney[1] was able to collect 46 cases from the literature where ligation of the ureter had been performed. Ten of these patients recovered without symptoms of any sort. Thirty of the patients recovered after a stormy convalescence, which required nephrec-

FIG. 2.—Dog 20. Five-day hydronephrosis after ligation of the ureter.

tomy in 7 of them. Six of these patients developed an infection of the hydronephrosis which required nephrectomy. As these cases are rather difficult to find in the literature and complete data was present only in the operative cases that required nephrectomy, the fate of the kidneys in the remaining 39 cases is problematical. From the standpoint of our subject, it is of great interest to know that about 15 per cent of the cases of accidental ligations of the ureter in patients resulted in infection of the

hydronephrosis. From the experimental standpoint a great deal of data is not available as to the spontaneous infection of hydro-

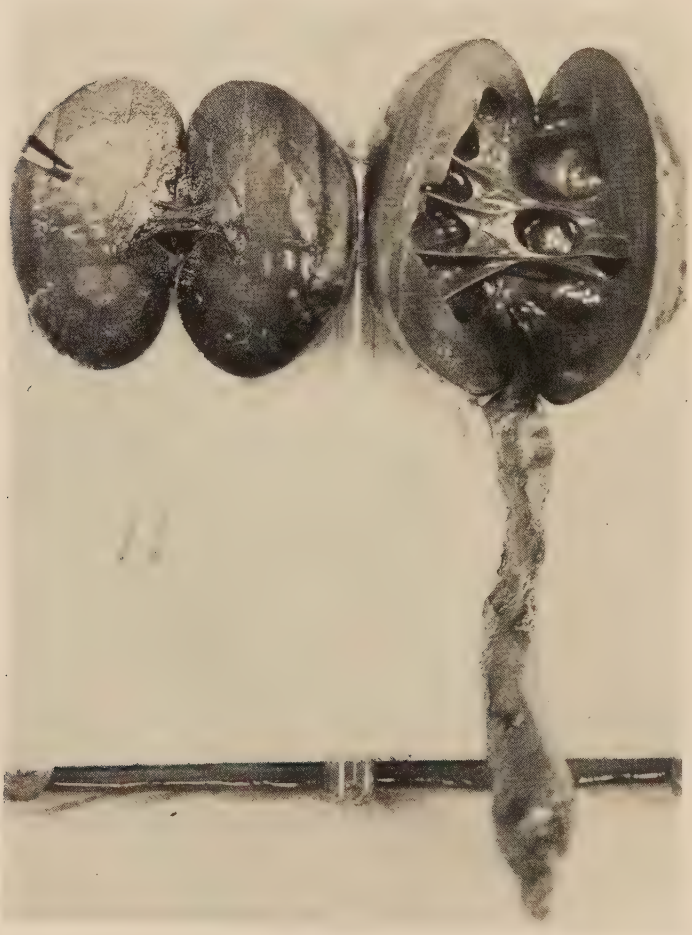

FIG. 3.—Dog 11. Six-day hydronephrosis after ligation of ureter and control kidney (*A*).

nephrosis after simple ligation. In some work of our own, where ligation and division of the ureter had been accompanied by division of the cord at different levels, we frequently had infec-

tion of the hydronephrosis when the operative wound over the spine became infected. We were able to ascertain that, as a rule, the organisms found in the pyonephrosis were those which were present in the wound infections of the back.

In the case of 5 other dogs which died after ligation of the ureter in a period varying from eight days to two months after the ligation, and in which no conditions had been introduced to favor infection of the urinary stream, spontaneously developing pyonephrosis was found in 2. One of these dogs died of distemper four weeks after ligation of the ureter. Fine bacilli commonly found in distemper animals were isolated from the hydronephrosis. In the other animal dying two months after ligation of the ureter, Gram-positive bacilli and Gram-negative cocci were found in the pyonephrosis. In contrast, an animal dying of pneumonia five weeks after ligation of the ureter had a sterile hydronephrosis.

These few clinical and experimental observations are of importance in that they show that the infection of a complete hydronephrosis occurs in a definite percentage of cases.

The experiments to be discussed in connection with this problem will consider contiguous intraperitoneal infections as well as the relation of the bowel and blood stream to infection of hydronephroses.

Ascending Infection of a Possible Route for Infection of a Hydronephrosis. The description of well-defined lymphatics in the wall of the ureter connecting the bladder and kidney has served as an argument to establish the premise that kidney infections may result from the infection traveling from the bladder to the kidney by way of the ureteral lymphatics. I believe this rarely happens and that the lymphatic drainage of the urinary tract is segmental, the bladder and lower ureter draining into the hypogastric glands and the upper ureter and pelvis of the kidney into glands along the vena cava near the hilum of the kidney.[2]

If, however, due to accidental ligation and division of the ureter in the course of a pelvic operation the stump of the ureter comes in contact with infectious material, the possibility of infection of the developing hydronephrosis arises. This infection

FIG. 4.—Dog 33. Six-day hydronephrosis after ligation of ureter and control kidney.

theoretically may involve the kidney by ascending infection through the ureteral lymphatics, by direct extension of the infection through the ureter or by involvement of the blood stream.

To establish experimental conditions which would enable us to evaluate these possibilities, 16 dogs, whose urine was known to be sterile, were used. In some of the animals a plastic exudate on the peritoneal surface of the bladder was induced by the colon bacillus. The ligated divided ureter became adherent to this plastic exudate on the bladder in several dogs. These dogs were sacrificed from four to twenty-three days after the experiment had started. The postmortem revealed a sterile hydronephrosis in 6 dogs, although a rather marked polymorphonuclear exudate was present in the periureteral tissue around the blood-vessels, frequently reaching to the subpelvic kidney fat. In 2 other dogs (six days and twenty-three days) Bacillus coli pyonephrosis had developed, though the opposite kidney urine, as well as the blood stream, was sterile. In these experiments the obvious inference is that the cut surface of the ureter coming in contact with infectious Bacillus coli exudate resulted in an ascending infection, which in 2 dogs resulted in a pyonephrosis.

In contrast to these experiments are those in which a piece of gauze saturated with Bacillus coli was wrapped around the undivided ureter. In these experiments very little or no evidence of involvement of the ureteral lymphatics was found and the urine in the kidney remained sterile.

In other experiments, where a pelvic abscess was formed about Bacillus coli impregnated gauze and the ligated cut end of the ureter was incorporated in this abscess, pyonephrosis was usually present after two to four weeks. In these animals it was difficult to say whether the infection had been caused by direct extension of the infection by continuity, or had spread to the kidney urine via the lymphatics, or was the result of a blood-stream infection. My impression is that lymphatic involvement was not responsible for the infection in this last group of animals.

In general, it may be said that a hydronephrosis developing because of a ligated, divided ureter may become infected via the

ureteral lymphatics if the cut end of the ureter is in contact with infectious material. I do not believe that this route is a common one or that it compares in frequency to infection of the urinary tract by other routes.

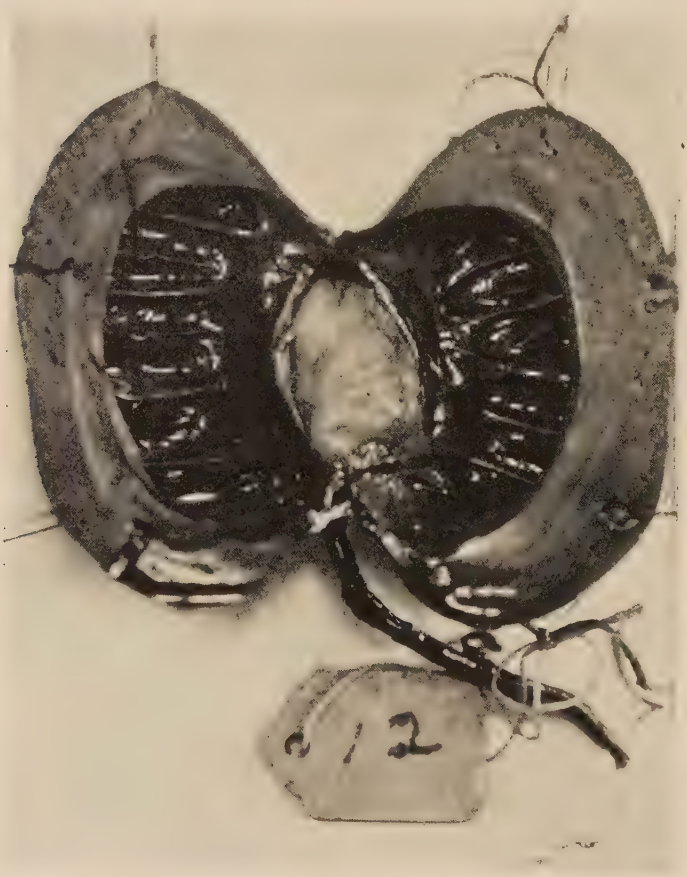

FIG. 5.—Dog 12. Eleven-day hydronephrosis after ligation of ureter.

THE RELATION OF THE BOWEL TO BACILLUS COLI INFECTION OF DEVELOPING HYDRONEPHROSES. There are several clinical and anatomical facts that point to the bowel as a possible source for Bacillus coli, which are so commonly found in urinary tract

infections. Bacillus coli has been isolated from the blood during the process of infectious diarrhea. Pyelitis in children has been noted most frequently with the incidence of summer diarrhea.

FIG. 6.—Dog DD. Two-weeks-hydronephrosis after ligation of ureter.

Several authors have mentioned the occurrence of constipation in pyelitis cases.

Anatomically, Franke[3] has described lymphatics connecting

the bowel and right kidney, and though he demonstrated these fine lymphatic connections in but 20 per cent of the bodies exam-

FIG. 7.—Dog BB. Thirty-eight-day hydronephrosis from ligated ureter. Death from respiratory disease. Sterile hydronephrosis.

ined, the theory of passage of organisms from the bowel to the right kidney has been rather firmly established in urological literature.

In reading Franke's articles on this subject, and in studying his drawings of injected lymphatics between the cecum and the right kidney capsule, the possibility of infection of the kidney by continuity from a diseased appendix or inflamed bowel adherent to the peritoneum over the kidney is obvious. It is questionable, however, how possible it is for infection to travel by the lymphatics from the bowel to the kidney if there be no gross lesion of the bowel or inflammatory process in the bowel which would cause adherence of the bowel to the kidney capsule. It is questionable because the lymphatic vessels between the bowel and kidney capsule are very small, relatively few in number and inconstant. The lymph flow from the intestine and appendix is to the mesenteric glands and not toward the kidney. Lastly, it is not definitely known that organisms leave the normal or slightly damaged bowel to gain access to the lymphatics other than the mesenteric glands. The presence of organisms in the mesenteric glands is by no means synonymous with kidney infection or blood-stream infection.

The striking work of Ravenel,[4] Calmette[5] and Griffith,[6] on the passage of tubercle bacilli and pigment through the normal bowel mucosa, makes it seem very probable by analogy that other bacteria might penetrate the normal mucosa of the bowel. Thiele and Embelton,[7] von Ficker[8] and others have reported experiments which seem to favor the point, while Opitz[9] and Neisser,[10] in carefully conducted work, throw great doubt upon it.

Author's Experiments on Dogs, Experiments were conducted on 37 dogs to study the possibility of the passage of bacteria, especially the colon bacillus, through the normal and pathological bowel wall to the mesenteric glands, to the blood stream or directly into the urinary tract through the lymphatics. A hydronephrosis was established on the right side by ligation of the ureter, to serve as a filter for any organisms reaching the urinary tract on that side. Controls were studied and experiments were conducted to ascertain the effect of the introduction of large amounts of actively growing Bacillus coli into the intes-

FIG. 8.—Dog EE. Four and one-half-month hydronephrosis from ligated ureter. Intravenous injection of B. coli three months and four months after ligation of ureter did not infect the hydronephrosis.

tinal tract of dogs during fat digestion, during periods of marked constipation or prolonged diarrhea and after obstruction or traumatism of the bowel.

FIG. 9.—Dog GG. Seventeen-months' sterile hydronephrosis from ligation of ureter. Intravenous injection of B. coli nine months after ligation did not infect the hydronephrosis.

Injection of Bacillus coli into the mesenteric glands and transplantation of impregnated Bacillus coli gauze into the mesenteric glands was carried out to attempt infection of the hydronephrosis via the lymphatics.

In obtaining material for culture at postmortem the abdomen was opened with sterile surgical technic. Bladder urine, right hydronephrotic urine and left kidney urine were taken for culture. Tissue culture of the mesenteric glands, kidney parenchyma and liver parenchyma were made by putting the tissue through a sterile Rosenow grinder and using the tissue emulsion in ascites bouillon and ascites blood for culture. Hearts' blood was used for blood culture. Culture of the urine by aspiration from the bladder was obtained by exploratory laparotomy in practically all of the experiments, and all dogs with organisms in their urinary stream were excluded.

As the details of the experiments have been published elsewhere, only the conclusions derived from them will be given:[11]

1. In control dogs with normal intestines the mesenteric glands contain bacteria in over 50 per cent and contain Bacillus coli in 33 per cent of the glands examined. These facts speak conclusively for the passage of organisms from the normal bowel to the mesenteric glands.

2. Injection of large amounts of actively growing Bacillus coli into the normal gastrointestinal tract of dogs was associated with the presence of Bacillus coli or a Bacillus coli-like organism in the urinary tract of 3 of 11 dogs. It is probable that these organisms reached the urinary tract by way of the blood stream. The lowered resistance of the animals afflicted with distemper seemed to predispose them to infection of the urinary tract.

3. Moderately prolonged constipation and diarrhea in dogs was not associated with relative increase of organisms in the mesenteric glands or with a urinary-tract infection.

4. Obstruction of the large bowel or traumatism of the bowel is associated with an increased bacterial content of the mesenteric glands, and urinary-tract infection occurred in two dogs.

5. No evidence was forthcoming that organisms reaching the

FIG. 10.—Patient died six days after bilateral ligation of the ureters during an operation for carcinoma of the uterus. At postmortem, both ureters were enormously distended. The right ureter measured 1.25 cm. in diameter and the left ureter 1 cm. in diameter. The walls of the ureter were thin, as though the dilatation had been acute. The renal pelves were markedly dilated and the urine contained in them was under high pressure. Pelvis of kidney outlined.

mesenteric glands from the bowel tended to involve the urinary stream by way of the lymphatics. It is probable that Franke's hypothesis in this connection would apply only to those instances where a diseased bowel or appendix was in contact with the peritoneum overlying the kidney. This explanation obviously would not include the great majority of kidney infections.

6. Cultures of liver tissue, bile and hearts' blood were uniformly sterile and led one to believe that organisms leaving the normal intestine do so in small numbers and are at the most but transient inhabitants of the blood or liver tissue.

7. The significance of the passage of intestinal organisms through normal as well as pathological bowel wall to the mesenteric glands cannot be overlooked. While it is undoubtedly true that these organisms reach the blood stream in but relatively small numbers and inconstantly, it is, nevertheless, possible that increased virulence of the organisms, lowered resistance of the host as well as actual lesions of the bowel wall would greatly increase the number of organisms reaching the blood stream. This conclusion would accord with the clinical relation between gastrointestinal lesions and urinary tract infections.

It is possible, therefore, that a developing hydronephrosis in man may be infected from organisms reaching the blood stream from the bowel.

Blood-stream Infection of Hydronephrosis. From the foregoing material it becomes apparent that blood-stream infections, from whatever source, are the most common means of entry of organisms into a developing hydronephrosis.

Many years ago Biedl and Kraus[12] and Brewer[13] demonstrated that organisms were rapidly secreted into the kidney urine after their introduction into the blood stream. To convince ourselves that Bacillus coli was thus easily secreted into the kidney urine after injection into the blood stream, 2 dogs were used. The right ureter was divided and a fine cannula placed into its proximal end. Cultures of the right kidney and bladder urine were taken for control. Two centimeters of a Bacillus coli suspension in salt solution were injected into the femoral vein,

and cultures were taken every few minutes from the right divided ureter as well as from the bladder urine. After one minute, four to six drops of right kidney urine grew seventeen to twenty

FIG. 11.—Patient died after prolonged labor. Autopsy revealed a bilateral hydronephrosis.

colonies of Bacillus coli. A decreasing number of colonies were present from the right kidney urine at intervals of four to thirty-five minutes after the intravenous injection of Bacillus coli. After thirty-five minutes only one colony was grown. This shows that Bacillus coli in the circulating blood is very rapidly secreted into the kidney urine, and that after thirty-five minutes it practically disappears from the kidney urine. This is of special interest in establishment of infectious lesions in the urinary tract, because without obstruction of the urinary outflow it is probable that the organisms are very rapidly eliminated in the urine and produce no lesions.

The effect of ligation of the ureter and development of a hydronephrosis on the kidney function has been carefully studied experimentally by Bradford,[14] Keith and Snowden,[15] Keith,[16] Johnson[17] and others. While the kidney still has some power of secretion at the end of two months, death results if urinary secretion is left entirely to a kidney which has been obstructed for only nineteen days (Johnson).[17] Hinman[18] found that a kidney obstructed for twenty-eight days did not regain any of its function. With these facts in mind a series of experiments were conducted in 10 dogs to ascertain whether it was possible to infect through the blood stream, a hydronephrosis due to complete obstruction of the ureter. In dogs having sterile urine at the time of ligation of the ureter, intravenous injection of 2 cc of actively growing Bacillus coli was carried out at intervals varying from one day to nine months after ligation of the ureter. It was found that infection of a hydronephrosis of one day, two days, fifteen days, twenty days and sixty days' standing was uniformly accomplished by intravenous injection of Bacillus coli. In contrast to these results were the failures to infect hydronephroses of three months, three and a half months, four and a half months, five months and nine months' duration by intravenous injection of Bacillus coli. From these experiments it seems justifiable to conclude that at some time between two and three months, after ligation and division of a ureter, the resulting hydronephrosis cannot be infected by way of the blood stream.

If by inference these results may be applied to the human, it is obvious that the real danger of infection of complete hydronephrosis is in the first three months of its development.

The accompanying illustrations indicate the degree of atrophy of the kidney parenchyma as well as the dilatation of the kidney pelvis following ligation of the ureter in the dog. The intrapelvic pressure taken in several of the hydronephroses of over three months' duration gave about 1.5 cm. pressure of mercury. The fluid in the hydronephrotic sac was straw-colored and slightly cloudy, and the following data was obtained from examination of the eighteen months' hydronephrotic fluid:

Appearance	Cloudy.
Color	Light amber.
Specific gravity	1011.
Reaction	Acid.
Serum	Albumin (plus, plus).
Casts	0.
Blood cells	0.
Leukocytes	Few.
Benzidin test for blood	0.
Crystalline elements	0.
Sugar	Very slight reduction.
Urea	Very little in comparison to urine.
Uric acid	2.20 mg. per 100 cc.
Creatinine	14 mg. per 100 cc.
Total nitrogen	164 mg. per 100 cc.
Non-protein nitrogen	104 mg. per 100 cc.

Compared to the figures for these substances in normal dog urine, they are present in very much less quantities, but, nevertheless, show many of the constituents characteristic of urine.

Conclusions. 1. Closed hydronephroses in dogs may be infected through the blood stream up to three months after the onset of their development.

2. Closed hydronephroses of more than three months' standing are no longer liable to infect through the blood stream.

3. Closed hydronephroses are rarely, if ever, infected through the lymphatics leading from the bowel to the kidney. Ascending ureteral lymphatic infection of hydronephroses is possible, but improbable.

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THE FULL THICKNESS SKIN GRAFT

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THERE seems to be little question that the ancient Hindus made free transplants of the full thickness of skin and included some of the underlying tissue, but their percentage of successes may not have been very high. The accepted technics and standards of today are not based on the Hindu method, but are rather an evolution, an early stage of which was recorded by J. Mason Warren in 1843. Its later developments have been chronicled by various surgeons down to and including J. Staige Davis, who has made valuable contributions to the technic and has given us one of the most comprehensive reviews and bibliographs on this subject. Another and more recent review is by Neuhof. From these latter we learn that the contributions of both Wolfe and Krause appeared chronologically rather late in this evolution.

In spite of all that has preceded, it still remains a surgical resource of which the profession at large is not making full use. The following account of our experience with this method of covering external surface defects is presented not as something new, but on the possibility that it may help to stimulate more interest in the procedure which in turn may hasten the elimination of certain malign influences that still cause our own results to fall far short of 100 per cent perfect.

Excluding army and veterans' hospital cases, it is based upon 106 autografts done in the past four years by Dr. Ellis Fischel, Dr. Earl C. Padgett and by the writer on patients in whom the latter was directly interested. A definite plan of technic was followed that required about a year to develop, and has not been materially changed since.

IN TWELVE CASES IN THIS SERIES 50 PER CENT OR MORE OF THE SURFACE AREAS WAS LOST. THE TABLE BELOW GIVES DETAILS OF THESE TWELVE CASES.

Source, location and reason for graft; age of patient.	Apparent cause and circumstances of loss.	Remarks.	Size of graft in sq. ins.	Per cent of graft lost.	Final disposition and results.
Abdomen to forehead, to replace delayed flap in infant, aged two mos.	Graft came off at first dressing with no evidence of ever having adhered; infection of bed due to contamination at operation and low resistance	After first of a series of operations for oblique facial cleft child had diphtheria in affected eye; later pus infection of the deformed lacrymal sac which was removed; of necessity the operating was resumed several weeks later; the conjunctiva became infected, the flap but partially adhered and an abscess developed in other side of neck	3	100	Raw area allowed to granulate; it will contract enough to allow the scar area to be entirely replaced by the pedicle of the flap when the latter is returned to forehead.
Abdomen to forehead, to replace delayed flap on young woman	Graft came off at first dressing with no evidence of ever having adhered; infection of bed due to operative contamination and low resistance	Graft was applied two weeks after a severe tonsillitis in a series of operations that could not well be interrupted	6	100	Thiersch grafted granulating area later, which "took" satisfactorily and made fairly satisfactory final result.
Abdomen into floor of mouth, through clean wound in neck, in an adult man	Most, if not all, of the graft was lost from maceration and infection, due to lack of ventilation and impossibility to carry out routine after-treatment	This graft was sutured around a wax form at normal tension and buried under scar in the floor of mouth that remained after hemiexcision of tongue; the incision was made from the neck and was presumably clean; Esser technic	3	100	Later, the tongue was again freed and an inlay graft "Thiersch," applied; final result, 50 per cent satisfactory.
Abdomen to forehead, to replace delayed flap in girl, aged fourteen yrs.	Infection of the graft which started in the epithelial surface after the graft had become adherent and before the original dressing was changed, possibly due to operative contamination and low resistance	This child has been through a long series of operations to replace cheek, floor of orbit, lower eyelid and half of nose and upper lip lost from noma; she is a subject of low resistance, and has reacted poorly after each operation and has had several infections during this period ranging from tonsillitis to hemolytic streptococcus of the conjunctiva; an earlier first dressing might have saved much of this graft	11	90	The granulations and remaining part of the derma were successfully covered with "Thiersch" grafts later; fairly satisfactory final result; at another time she had a successful full-thickness graft applied to the other side of forehead.
Abdomen to cheek, to replace burn scar on young man	Graft appeared perfect at first dressing; two days later when pressure dressing was left off to avoid its contamination with saliva, flap became blue and ultimately died from blood stasis in spite of oft-repeated scarification and citrate-of-soda pack	Where this pack rested on the body of the mandible some of the graft was preserved; it was the experience with this graft that suggested the necessity for the longer retention of the pressure dressing	12	70	Was regrafted, full-thickness graft after slicing off granulation 36 days later; this second graft contained less than 5 square inches of skin.

Abdomen to front of the neck, to release burn scar on girl, aged fourteen years	Graft became attached, but on the day after operation certain areas appeared to be without elasticity; on cutting in it was found the skin was dead; the slough was gradually thrown off (Fig. 5); necrosis apparently due to too firm pressure	This was one of the early cases in the development of the pressure dressing; the sponges were bandaged in so tight that the face was congested; pressure over the upper part of larynx is particularly apt to kill the graft in this situation	32	70	Regrafted at a later date, when raw areas had scared up.
Abdomen to cheek, nose, lower lid and upper lip, to replace burn scar on infant, aged two years	Graft was compressed with a rubber bath sponge; death seemed to be due to maceration caused by lack of ventilation and subsequent infection	This was one of the several grafts which were covered by a more or less impervious dressing, all of which macerated to a greater or less extent	2	70	Area covered with a pedicle flap from forehead at a later operation.
Abdomen to lower eyelid, cheek and side of nose on retarded child, aged seventeen years, due to a congenital infection	Graft adhered throughout, but about half was lost apparently from superficial infection becoming active in the second week	In this case the vesicated areas were painted once a day with 1 per cent silver nitrate in alcohol, and it was thought this might possibly have helped to damage the epithelium; the graft contracted somewhat less than one-half its original area	3	50	Eyelid later restored with pedicle flap graft; the graft itself is a little glazed.
Abdomen to cheek, to repiace burn scar in a boy, aged six years	About one-half the graft became necrotic, roughly corresponding in area to the body and ramus of jaw and zygoma, but it was firmly attached at first dressing	Slough was finally thrown off, in places leaving granulations, in others a stippled base; the behaving and distribution of dead skin seemed to indicate that too much pressure had been used; note that in Case V of this series, where a very loose dressing was used, that part of the graft overlying the bone was the only part saved	12	50	Scarred up and made a fairly good final result with all distortion relieved; the scars are red and rough, but time will largely correct this.
Abdomen to front of the neck, to replace burn scar on girl, aged nineteen years	At first dressing, ninth day, a necrotic area was discovered over the upper part of the thyroid, while the "take" lower down and in the submental and submaxillary regions was perfect; too great pressure and too much movement	On cutting into dead area two things were observed: (1) That part of the dead graft in front of the pomum adami and thyrohyoid membrane was not adherent, but surrounding this was a ring of dead graft that was adherent to the deeper tissue; (2) that a narrow bridge of live skin spanned the central part of this unattached and otherwise dead area	7	50	Allowed to scar; provision for loss had been planned for in the cutting and suturing the graft.
Abdomen to the palm of little finger, for burn scar in a young woman	Implanted partly on scar; considerable superficial loss and contraction—cause not very evident	.....	1	50	Some flexion of the finger remained.
Abdomen to front of the neck, to release burn scar on child, aged six years	Dressed 5 days after operation; much of graft macerated, due to poor ventilation and infection	In this case the bandages over the marine sponge were daubed with vaseline to prevent possibility of vomit getting into sponge	22	50	Additional grafts added after healing.

NOTE.—These operations were performed at the Barnes Hospital, the St. Louis Children's Hospital and the St. Louis Mullanphy Hospital. This tabulation does not include quite all of our cases, for in many instances the skin graft was but one incident in an operation, and has not always been separately indexed.

All negative statements that may follow are to be taken as referring only to the cases in this series and not to imply that positive results might not be obtained by some other technic or under some other circumstances.

Dissatisfaction with the ultimate results of the "Thiersch" graft when used on skin surfaces was the driving motive. The work as started was based on two premises: First, that if the transplanted skin were held at its normal tension or at slightly plus tension the cut ends of the cutaneous vessels would be held open and would more quickly take up a blood supply; the second, that as each particular part takes its blood supply from the immediately subjacent tissue the only logical limit to the size of a graft would be the amount of operating and hemorrhage the patient could stand. Subsequent experience justified both of these conclusions. A very free arterial blood supply has been demonstrated by scarifying a graft forty-eight hours after implantation (Figs. 1 and 2), while numerous subsequent observations have proven that a graft not on tension is uncertain about acquiring a blood supply (Fig. 3).

Very soon after these two hypotheses were put into practice, it became evident that there were additional factors of almost equal importance that still remained to be catalogued. Certain of these have since been identified, but not without some heart-breaking experiences and the indulgence in considerable worry; others have so far baffled our efforts. The first of these stumbling-blocks was a fact long ago recognized in dealing with large, thin flaps; that it is one thing to have an arterial blood supply and quite another to have an adequate venous return. In the first several cases the grafts were applied to the bridge of the nose or to the forehead (Fig. 2). In the former the tension of the

sutures drawing the graft over a curved, firm surface, and in the latter the pressure of the bandage that held the dressing, both helped to limit the amount of blood that could stagnate in the skin while the new venous return was being established. It

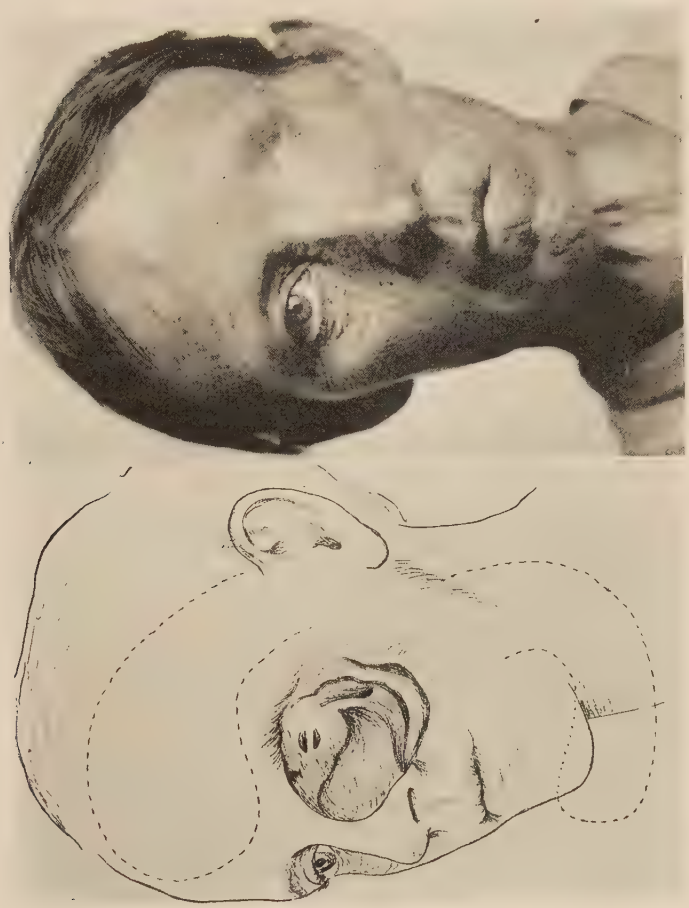


FIG. 1.—Shows a full thickness graft on the forehead used to replace a flap that covered the cheek or orbit defect. On the third day after operation, because the graft was found to be dusky, the epidermis was incised to relieve the congestion and this produced a very free flow of bright red blood.

was not until the attempt was made to place a large graft on the cheek that we were forced to conclude that in the previous cases good luck had outrun calculation. In the particular case in point the retaining dressings were removed at the end of two

days for fear mouth secretions might seep under them and the newly adherent graft was left exposed. Within a few hours the pale pink skin became deeply blue and repeated scarifications, carried on night and day, and citrate of soda-packs failed to

FIG. 2.—Shows boy with burnt face. The end of the nose is abbreviated and there is an ectropion of the nostrils which caused the nostril hair to grow on the outer surface of the ala. To correct this a transverse incision was made across the dorsum from cheek to cheek and the scarred superficial tissue was undermined and rolled downward until the ala and tip were restored. The resulting diamond-shaped defect was filled by a full thickness graft that was sutured in under tension. This was the first case of this series and the ease with which the result was obtained led to the erroneous conclusion that suturing in the graft at normal tension was all that was needed to get 90 per cent satisfactory takes.

save us from the painful necessity of explaining to the patient the loss of $\frac{7}{10}$ of a 3 x 4 inch graft. Three months later, after shaving off the granulations, another graft was applied, but this time it was protected by dressings held in place with a pressure

FIG. 3.—Shows defect filled with a $3\frac{1}{2}$ x 8 inch full thickness graft from the abdomen in a man aged fifty-six years. This graft was cut by a pattern that was the size of the defect, but instead of the graft contracting, as is usually the case, it stretched so that when it was sutured into the defect it was quite loose. (Operated on March 21, 1923.) In (a) taken thirteen days after placing the graft the imperfect "take" is already apparent. In (b) taken twenty-two days after operation the dead skin has been thrown off and the granulating areas are evident. (c) Taken one year later shows the surface healed partly by scar. The cross lines show the areas into which sensation has been restored, but in the scarred region it has not returned.

bandage. That part of the graft survived which lay over a wax form in the hollow of the cheek which furnished counter-pressure (Fig. 4). Based on this experience a marine sponge pressure-dressing was evolved which, if carefully applied and

FIG. 4.—Shows a full thickness skin graft on the forehead that replaced a "delayed flap" used to reconstruct the nose. The central part of this flap was thicker than the outer part, which left a deeper depression in the center of the resulting defect. A simple gauze pad was bandaged over the graft and this did not compensate for the lack or counter pressure in the central part. It will be seen that the central part of the graft where the pressure was less has died as the result of venous stasis.

made of sponges of proper quality, will maintain an even pressure over an irregular surface or one without underlying bony counter-support. Further use of this pressure-dressing demonstrated an unforeseen virtue and an inherent danger. It permits of the

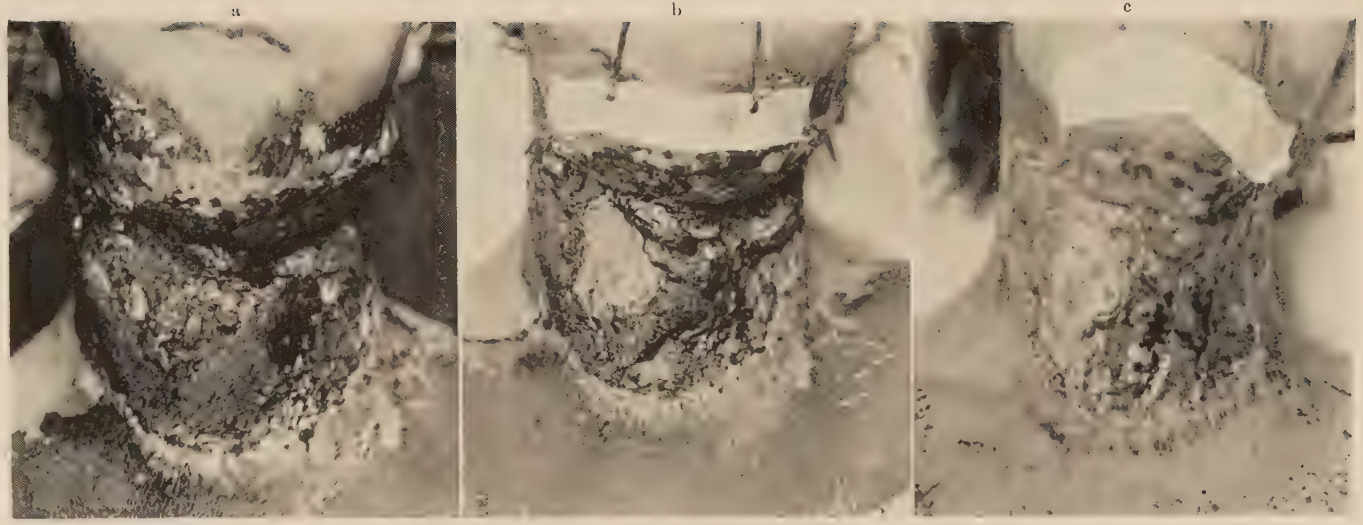


FIG. 5. (a, b, c).—Show three stages of a graft, 70 per cent of which was lost from excessive pressure. This was one of the very earliest cases in which the sponge pressure dressing was used and it was put on so tight that the face was dusky from venous congestion. (a) The first picture, taken the day after the original dressing was changed, shows much of the graft dark and inelastic but adherent. In (b), taken two days later, the most superficial layer of epithelium has peeled off, showing a patch of pink, live skin. In (c), taken twenty-two days after operation, the dead part of the graft has been thrown off, leaving a patch of live skin and a granulating surface. The submaxillary and submental graft, which was not subjected to so great direct or counter pressure, survived almost intact.

application of the graft over a freshly made raw surface, such as results from the removal of a scar, without tying any but large vessels, but if the pressure is too great, especially over a bony prominence, such as the outer part of the supraorbital ridge or dorsal surface of the middle metacarpal bone, it can kill the compressed area by ischemia (Fig. 5). Maintenance of the proper pressure for four or five days will prevent the graft from

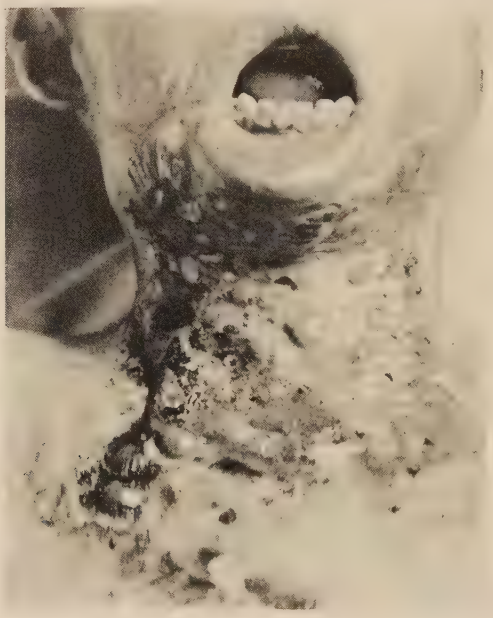


FIG. 6.—Shows the blebs that form after the too early removal of the pressure dressing. These blebs may form under the pressure dressing, but are not apt to be so extensive nor so troublesome.

dying from engorgement, but its early discontinuance favors the formation of blebs, which latter may lead to another catastrophe (Fig. 6). The blebs are apt to become infected, sometimes, at least, from organism contained in the skin. If this infection is not controlled the resulting ulceration may not stop until it has eaten through the full thickness of the skin. This ulceration seriously marred a number of our earlier final results, and

FIG. 7.—Shows a complete syndactylism in a five-year-old boy in which the two outer clefts were lined with full thickness grafts and the two inner with "Thiersch" grafts. All were sutured in place, the whole operation requiring one hour and ten minutes. The clefts were cut as deep as could be without dividing the transverse metacarpal ligament, but the grafts contracted so that it was advisable to again deepen the clefts and regraft approximately eight months later. At this time the full thickness grafts had contracted considerably less than had the "Thiersch."

in many of our later cases we were only somewhat less bothered by it. Dr. Martin Engman, while seeing one of these cases in consultation, advised painting the infected base of the bleb with a 1 per cent silver nitrate solution. We have since continued its

FIG. 8.—Case of complete syndactylism of the foot. Here part of the clefts were lined with "Thiersch" grafts and part with full thickness. Note how, after freeing the toes, the foot flattened out to form a usable member. In this instance the subsequent contraction, though quite as great as in the preceding, did not interfere with function.

use, and I think it greatly helps to control this ulceration. Also to help control this infection and to prevent the dressing from sticking, a layer of gauze impregnated with 3 per cent xeroform ointment is laid on the graft under the pressure dressings. The

use of this ointment is continued as long as dressings are deemed necessary, and it is certain that our later results are much less marred by scars resulting from patchy destruction of the epidermis than are our earliest cases; however, this source of error has not yet been entirely eliminated. Very recently, to control this superficial infection, we have in several cases used a 2 per cent boric acid pack under an impervious dressing, apparently with somewhat more promising results (Fig. 20).

FIG. 9.—Shows the partial result that followed the removal of the scar, straightening the finger and covering the raw surface with a full thickness graft in the case of a contracted burn scar. No allowance was made for the subsequent contraction of the scar surrounding the graft and the result can be rated as only a partial success.

Blood-clots of any size will cause the death of the overlying skin. It is impracticable to tie all or sometimes any considerable number of the bleeders after the removal of a burn scar, but properly graduated sponge pressure following appropriate puncturing of the graft with a fine leather punch, as advocated by Davis, or with a knife, and carefully squeezing out all clots

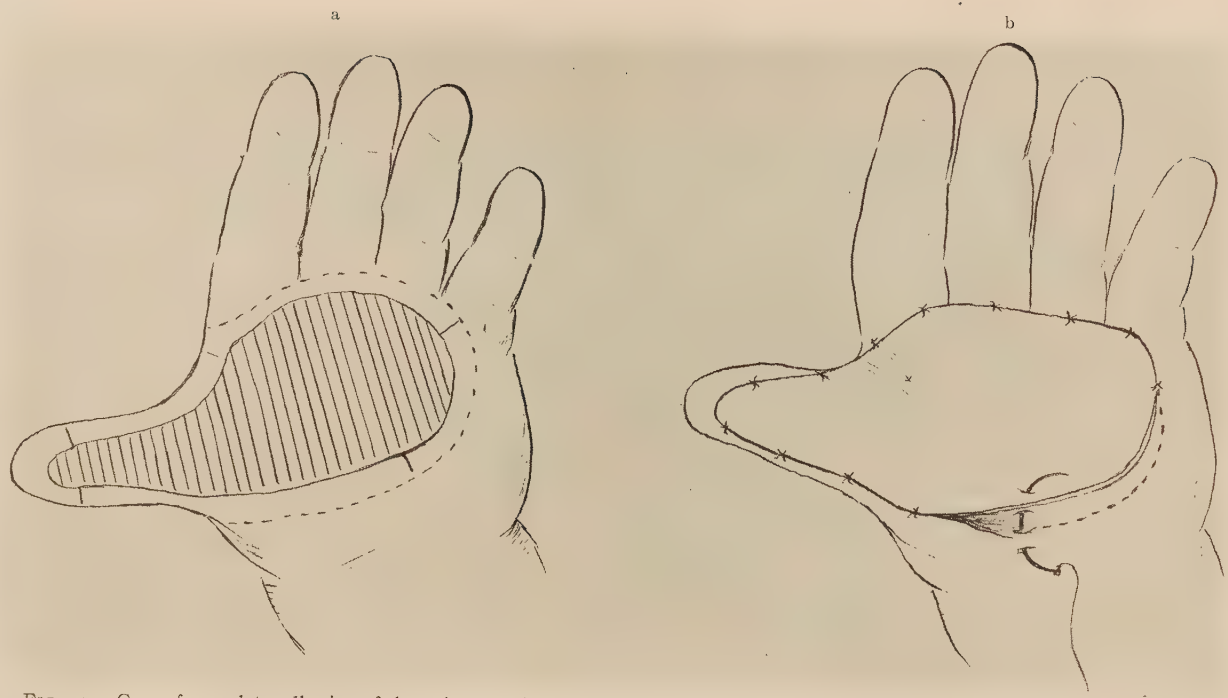


FIG. 10.– Case of complete adhesion of the palmar surface of the thumb to the palm of the hand following a burn. The striped area in the right hand figure shows the extent of the raw area that resulted from the freeing of the thumb; the outer lines indicate the extent to which the surrounding skin was undermined. In suturing on the graft these undermined edges were turned outward, skin surface to skin surface, so that the area of the raw surface was increased by almost twice the area of the undermining. Each suture was passed first through the edge of graft then through the everted skin edge, and then took a turn through the palmar skin along a line corresponding to the edge of the graft and everted skin. This causes an elevation of the edge of the graft which might slough from the greater pressure to which it is exposed if an even thickness of gauze and sponge is simply bandaged over the whole. Compensation can be made by filling the depressed central part by several layers of xeroform gauze cut to accurately fit within the elevated edges. When the sutures are removed after following this plan, at first the grafted area is surrounded by a ridge, the outer surface of which is formed by the everted skin, the inner surface by the graft. This subsequently flattens out. If any did remain it could be removed after one was sure that the redundant skin was not needed. In cutting the graft for this plan it is made the same size as the raw area when the undermined edges are everted.

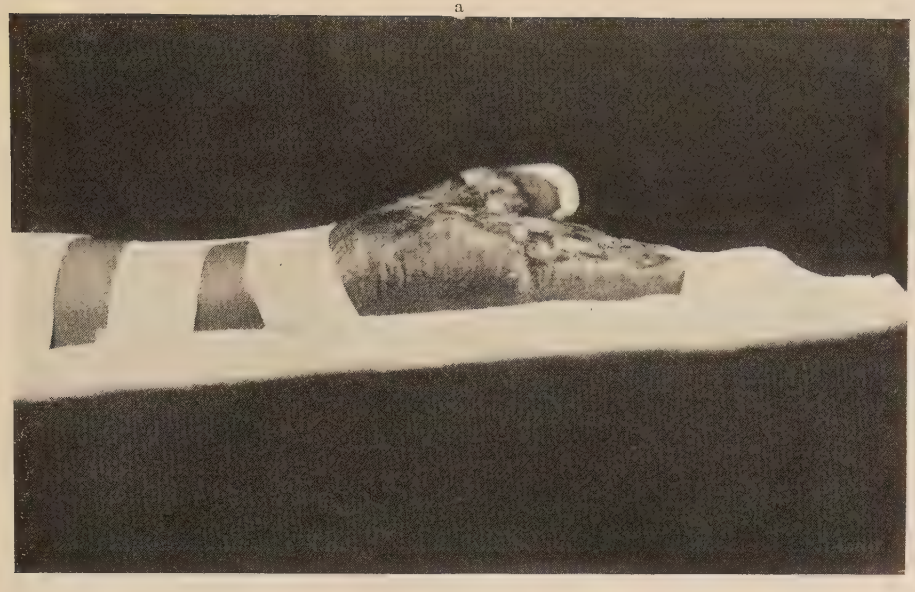
before applying the pressure dressing, will do much to eliminate this source of disaster. A "Thiersch" graft can be wrapped around a wax form and buried in the tissues with every expectation of a perfect "take," but our observation leads to the belief

FIG. 11.—Shows case illustrated in Fig. 10. To the left is seen the thumb united to the palm while the photo on the right shows the final condition. Note that the ridges of superabundant skin have disappeared but that the thumb is completely freed.

that the full thickness graft requires some ventilation during the first few days after it has been transplanted. This conclusion is based on the following experiences: Several times a rubber bath sponge was used in place of a marine sponge; in one case the outside dressings were all covered thickly with vaseline, to

FIG. 12.—The photograph above shows a child with both arms bound down by scars in and below the axillæ while the lower one shows them released after incising the scars, abducting the arms, and covering the raw surfaces with full thickness grafts. At the first operation both arms were freed. In the right axilla a full thickness graft from the mother, whose blood matched with the child's, was implanted while on the left a graft from the child was used. The subsequent behavior of these grafts is detailed in the text. At a later operation the right axilla was regrafted from the child.

a



b

FIG. 13.—One of the great advantages of the full thickness graft over the pedicle flap in the restoration of the burnt hand is that the hand can be conveniently splinted while the free-skin graft is healing in place. It is seldom that the skin over the palmar surface of the distal phalanx is involved. After removing the palmar scar, undermining the edges, and suturing in the graft after the plan shown in Fig. 10, a folded piece of $\frac{3}{8}$-inch mesh sterile "Hardware cloth" is appropriately padded and the hand is held extended by strips of adhesive plaster that pass over the distal segment of each finger and which are tied through the meshes of the splint. The fingers should at the same time be somewhat separated and the thumb completely abducted. One or two adhesive straps around the forearm complete the fixation after which the usual sponge pressure dressing is bandaged in place. It is well to reinforce this wire splint by dandaging a light board on the dorsal surface after the dressings are applied. We had one graft damaged some days after operation by a fall in which the splint bent. (a) Shows this wire splint applied. The extensor tendons may have become fixed in a burn scar of the dorsum and these can be released when the scar is removed so that the fingers can be flexed. The graft should be patterned so that it will cover the defect with the hand flexed and the hand should be splinted in this position, shown in (b), and this should be continued for some weeks.

prevent the possibility of vomit soaking into the marine sponge; in another case the full thickness graft was sutured around a wax form under normal tension and buried in the floor of the mouth through an external incision, and in still another a very soft, old sponge was put on with such pressure that it made an almost impervious casing. In the above cases much or all of the grafts macerated and a considerable part of each graft was lost. There may be still other causes that produce local areas of "death" of the graft, and upon this subject we are still seeking light.

The shrinkage of the graft bears no proportion to the amount of contraction that occurs in a "Thiersch" graft. If the epithelium is preserved the contraction will be not more than would occur in the healing of a pedicle flap of the same size. If there is more than a very superficial loss of the epithelium the area from which this loss occurs may contract one-half or more (Fig. 19). In some localities, as the forehead, this normal contraction need not be considered, in others compensation must be provided beforehand or made subsequently. This can be done in some areas by doubling back the skin bordering the defect at the time of operation (Figs. 10 and 11), in other instances an additional graft or a flap has been inserted to release the tissues displaced by the contraction (Figs. 17–19). On the hand, or axilla, splints used for three weeks will help control contraction (Fig. 13), and the lips or eyelids can be supported either by uniting the corresponding fellows, by suturing newly made raw surfaces or by suspension sutures (Fig. 19).

The final color of the graft is a thing that we cannot foretell, except that in a general way the darker the complexion the darker is apt to be the tan that will come in the graft. Normally, the abdominal skin has a yellow tinge, but this does not account for the tanning that occurs to a greater or less extent in the majority of full thickness grafts. On "white" negroes this tanning may be very dark. Usually the tan is evenly distributed, but sometimes it is blotchy (Fig. 16, *c-d*); where the graft does not tan it is apt to be pale or white (Fig. 16, *a-b*). In some cases the

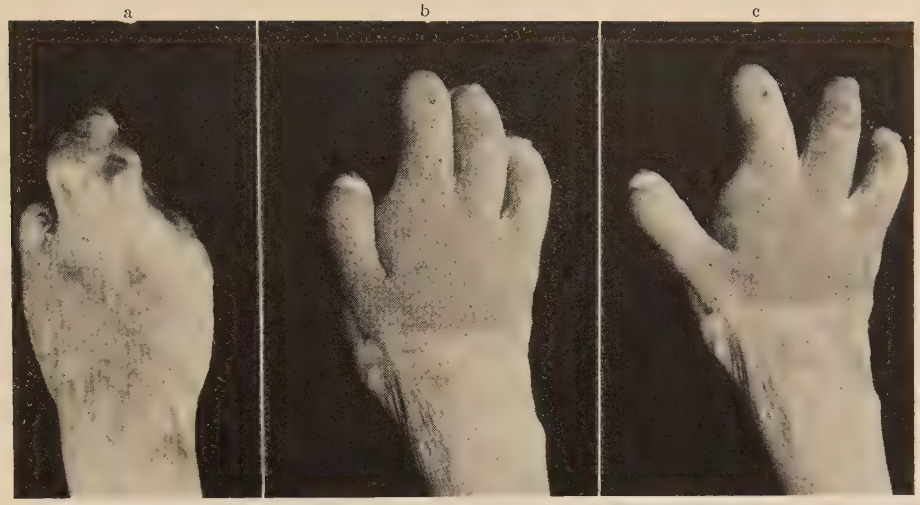


FIG. 14.—The photograph to the left shows a hand burnt on all surfaces with the fingers scarred into one mass resulting from a burn that involved the dorsum and interdigital spaces. The dorsal skin was restored by removing the scar and planting the hand and fingers in pockets tunneled just under the skin of the lower abdomen. Later the interdigital spaces were epitheliated by full thickness grafts. Figure (b) shows the restoration, figure (c) shows the ability to abduct the fingers.

FIG. 15.—Shows the rough scar resulting from a burn sustained years previously. The whole scar was dissected off and the defect covered with one full thickness graft from the abdomen. Figure (b) shows the graft which "took" throughout. It is of good color and texture.

transplanted skin will become more pink than it was in its natural position and match the face skin fairly well; tanning appears somewhat late.

FIG. 16.—(a) Shows a lady on whom the nose and upper lip were remade from a flap taken from the neck and the tissue behind the ear. (b) This was replaced with a full thickness skin graft from the abdomen. In this case the "take" and texture were very good but the skin grafted is very much whiter than that of the neck. (c) Shows a young woman who, on account of a recurrent carcinoma, had part of cheek and upper lip reconstructed from a delayed flap taken from the forehead. The forehead defect was filled by a full thickness graft from the abdomen, satisfactory in "take" and texture but which has become very darkly tanned. The girl is naturally of a dark swarthy complexion (d).

In a general way, age has been an influencing factor. As a rule, the younger the patient the more certain is the graft to "take" completely and the more natural will be the appearance. However, in a man, aged seventy-nine years, who had a $4\frac{1}{2}$ x 6 inch graft transplanted from the abdomen to the forehead,

FIG. 10

the color and texture were so perfect that it could not be detected by any casual observation. The youngest in this series were two infants grafted for complete syndactylism at the age of one month each. In the adult, and even in young children, these

FIG. 17.—(a) Shows the appearance of a child shown in Fig. 18 after 50 per cent of the first graft had healed in place. (b) Shows the case after a second strip of skin had been recently grafted above the upper edge of the first. The completion of this case is shown in Fig. 18 (b).

grafts are as resistant to contamination at operation as any healthy tissue, but should infection occur it is apt to be very destructive to the graft itself. At many of these operations it is practically impossible to keep the field clean. In the infant

FIG. 18.—Shows a child who as a result of severe burns had the mucosa of the lower lip attached to the chest 1 inch below the upper border of the sternum. It required three separate operations to obtain the result shown in (b). At each of these operations full thickness grafts from the abdomen or thigh were implanted. A total of 50 square inches of skin was transplanted, eighty per cent of which "took"—color good.

and young child a pus infection may very rapidly destroy large parts of an already adherent graft within the first few days

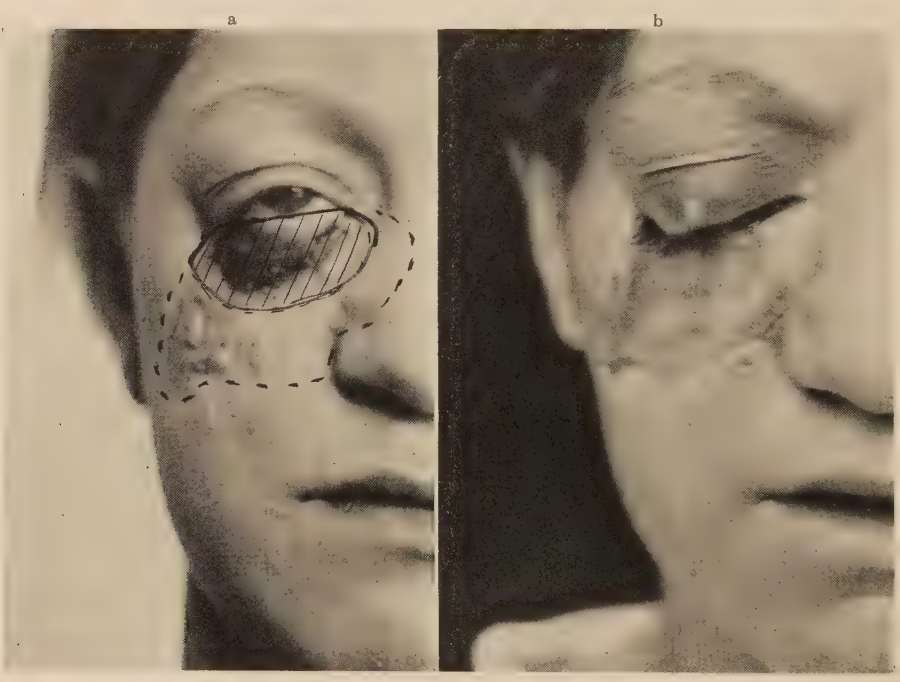


FIG. 19.—Shows a child on whom a scar of the lower lid and cheek, due to congenital infection, was replaced by a full thickness skin graft from the abdomen. In order to hold the denuded lower lid on tension while the graft was healing in place, a small raw surface was made in the middle of the upper lid just above the tarsal border and a projecting tab of graft was made to jump the palpebral fissure, tarsal border and eyelashes to be planted on the upper lid. This strip of graft "took" perfectly and can be seen in the figure. The part included within the dotted outline in (a) shows the amount of scar actually removed while the everted mucosa above this was undermined and turned upward, thus increasing the raw surface to be grafted to the extent shown in the striped area. A superficial ulceration caused the subsequent loss of some epithelium with a resulting excessive contraction of the graft, which necessitated the subsequent addition of a pedicle flap from the forehead to the lower lid. Finally the skin bridge across the tarsal fissure was cut and the piece removed from the upper lid.

after operation, and this is most apt to occur when the original dressing has not yet been disturbed. It has, however, been our observation that grafts on the infant and young child acquire

FIG. 20.—(a) Taken on the twelfth day after operation, shows a full thickness graft that was practically perfect at that date. Up to this time the graft had been dressed with xeroform ointment on the gauze. The ointment was left off and a dry dressing was applied for but twenty-four hours. (b) Shows the same graft one week later. A superficial ulceration has spread over about one-half the surface and in two small areas has gone completely through the epidermic layer. A 2 per cent boric acid, moist pack, frequently changed, was used which controlled this superficial infection. The xeroform or the boric acid may be of some help, but what really accomplished the result was the ointment or the wet pack which prevents the formation of crusts that harbor bacteria. The discolored appearance in (a) is due to xeroform, which turns dark after a few days.

FIG. 21

FIG. 21.—Histology of Auto Grafts of Skin.—In making a study of the histology of skin grafts Dr. Earl C. Padgett found that the most complete recent report was from "Transplantation of Tissues," by Harold Neuhof, D. Appleton & Co., 1923, and is taken to be a *résumé* of the present ideas in regard to microscopic appearance of transplanted skin. He states that within five or six hours after transplantation there is an exudate of fibrin from the wound fixing the transplant in place. This fibrin layer soon becomes infiltrated with leukocytes and fibroblasts and disappears gradually to be replaced by a richly vascular connective tissue containing many round cells. The granulation tissue has changed into an organized membrane at about fourteen days. Within a few hours after the graft has been applied, leukocytes of the fibrin layer migrate into its interstices and are also to be found in the lumina of its empty bloodvessels. Most of the bloodvessels in the transplant degenerate. By injection experiments vessels have been demonstrated in cutis grafts on the third day. The newly built vessels arise by a budding of the capillaries in the fibrin layer, and the buds not infrequently extend directly into the vessels of the graft. Degenerative processes set in so that on the third or fourth day the epidermis with the upper rate layer of the skin are lost. The degenerative processes are pronounced at an early stage so that there is a vacuolization of the surface of the graft, hand in hand with the degenerative processes, regeneration goes on and it is usually so energetic that in six or eight days the entire transplant is covered with new epithelium. The degeneration extends beyond the epithelium to the cutis. The entire cutis is replaced by granulation tissue which in time shows a decrease in cells and bloodvessels and an increase in fibrous tissue. According to this, histologic examination established viability only, and this solely in the juxta-epithelial layer of the graft. The histologic changes in the whole thickness grafts do not differ materially from those occurring in the "Thiersch" grafts. The epithelium is slower to regenerate in full thickness grafts by a few days. Doctor Padgett's study of sections from our own cases does not show as much degeneration of surface epithelium nor of the cutis and corium. Sections taken from grafts which have good color show only a very superficial degenerative change in the outermost layer of epithelial cells, and when this layer is removed it leaves a skin which is a little more pink but otherwise normal in appearance, and microscopically the squamous cells appear a little edematous and swollen. Evidently only the most superficial epithelial cells degenerate on good grafts. He does not believe that our sections show either as much degeneration of the squamous epithelial layer or of the cutis and corium as is usually described. The older grafts on inspection look rather normal in texture but a very pale tan with little suggestion of the normal pink of the surrounding skin. Histologic studies were made of approximately twenty grafts of this series varying from eight days to two years after implantation. The most noticeable change that occurs in the microscopic anatomy of an old full-thickness graft is a smoothing of the surface best appreciated when a section of the graft is compared with a section taken from the site that furnished the graft. The papillary layer of the epithelium appears to be about normal except that it follows the flat contour of the surface. Elastic tissue appears to be present in about normal quantity. Fig. 21c, is the abdominal skin taken from alongside of the scar which resulted from removing the graft shown in Fig. b. Note the surface irregularities. Fig. b shows a section of the graft twenty-two months after it was implanted in the forehead, where it "took" without any hesitation or any evident loss of epithelium. In Fig. a the papillary layer is brought out with more contrast. This is a section from a graft on the forehead of a six-year old boy done eight months previously, which has the same general appearance as the graft b. Note the hair follicle and gland remnant and the same absence of the normal surface irregularities that is to be observed in b.

an immunity to infection earlier than do the grafts on the adult. On the latter the graft seems to be more apt to weather the first week, but is very susceptible to superficial infections occurring under the scarf skin which become manifest in the second or early part of the third week. Such infections can in a few days destroy or very much cripple a previously perfect graft (Fig. 20). In one three-year-old child, who at the time was not under close observation, a destructive ulceration attacked a forehead graft in the third week after operation.

On two cases a graft was transplanted from the mother to the child; in one the blood matched and in the other it did not. In each it was controlled by an autograft made at the same time, and in each, at the end of the first week, the homograft appeared to be a perfect "take," even smoother than the autograft. In the second and third weeks, however, the homografts macerated and were thrown off while the autografts made good (Fig. 12). This is contrary to some reported experiences. Dr. M. T. Burroughs suggested to us that difference in age between mother and child may have been a factor in the failure. John Staige Davis, J. C. Masson,[1] H. K. Shawan,[2] and others have reported success with the homograft, while R. Minervini[3] used the skin of non-diseased stillborn babies, and his reported success in this might have some relation to our observation that the younger the child the more satisfactory the "take." It would be a great blessing in certain burn cases of young children if the skin could be obtained from some other source.

The individual grafts varied from $1\frac{3}{4}$ to 42 square inches. We have found that the transplantation of about 40 square inches of skin approaches the practicable limits of the operation, especially when this step is just a part of one operation. The following tables give a *résumé* of our work:

[1] Jour. Am. Med. Assn., June 1, 1918.

[2] Am. Jour. Med. Sci., April, 1919.

[3] Transactions of the Medical Congress, Jour. Am. Med. Assn., September 6, 1913.

Indications.	Number.	Average per cent of success.
Replace delayed flap	34	79.70
Replace immediate flap	2	90.0
Release or replace burn scar	57	77.87
Replace eyebrow	7	87½
Release ear	3	66⅔
Web fingers	4	76½
Web toes	1	90.0
Release Dupuytren's	1	90.0
Scar	1	75.0
Replace nevus	1	80.0
Luetic scar	2	67½
Release tongue	1	0
Replace scalp	1	90.0

Location of graft.	Number.	Average per cent of success.
Forehead	38	81.32
Cheek	8	58.0
Eyelid	4	85.0
Dorsum of nose	2	85.0
Neck and chin	12	68.0
Hands, dorsum	10	83½
Hands, palm	9	83⅓
Finger clefts	5	76.0
Axilla	3	80.0
Popliteal space	2	70.0
Floor of mouth	1	0
Toe clefts	1	90.0
Arm	1	80.0
Neck	3	85.0
Eyebrow	7	87½
Ear	2	55.0
Forehead and scalp	3	86⅔

Tissue on which graft was bedded.	Number.	Average per cent of success.
Periosteum of forehead	31	78.70
Subcutaneous tissue	13	76.53
Subcutaneous and burn scar	57	77.28
Heavy scar	1	50.0
Partly on tendon	5	90.0
Partly on cut bone	2	90.0
Luetic scar	2	62½
Tissues below floor of mouth	1	0
On a granulating surface	1	80.0

Number of graft operations	106.0
Average per cent of success	77.41 per cent
Number showing 90 per cent success	58.0
Average size of grafts or better	8.43 square inches

These estimates are, of course, somewhat approximate, though all grafts were cut from tin-foil patterns. As a margin of safety in making estimates, no graft was given credit for more than 90 per cent "take." Where provision for shrinkage or loss had been made a part of the operation, then the final result, not the amount lost, was taken as the basis in making the final estimate of success. Many of these grafts took perfectly, and except for the bordering scar and a slight tanning and shiny appearance, the skin substitution could hardly be detected. In most localities a graft can be so placed as to allow for 25 per cent shrinkage.

Most of the grafts were taken from the abdomen, but some of necessity came from the thighs or sides, and two were taken from the back. No attention was paid to matching the direction of the cleavage line of the skin, though possibly this might in some way have some influence. It seemed that the thin skin "takes" more kindly than thick.

Seven of these grafts were taken from the scalp to replace lost eyebrows, and we have in some cases, not included in the above series, transplanted free grafts from the eyebrow to replace eyelashes. In replacing eyebrows the scalp rather than pubic skin has been used, for the reason that the hair on the former grows more closely and an accurate selection can be made as to the direction in which the hairs grow. It is true that the hair on a scalp graft must be trimmed at intervals, but that might be true of pubic hair, and the plan of growth of the latter resembles eyebrow less than does the scalp hair. Law, of Minneapolis, has mentioned one case in which an otherwise successful pubic graft had to be removed on account of embarrassment arising from the self-consciousness of the patient.

Its Place in Surgery. The four most appropriate uses we have found for the free full thickness skin graft are the release of scars on the neck and axillæ, the replacing of burn scars on the hands, neck and forearms, to replace flaps that have been taken to repair other defects and to furnish skin to line clefts in congenital or acquired syndactylism.

In these uses it can be compared with the pedicle flap and the

"Thiersch" graft about as follows: The former, when properly handled, is more certain of complete success, and if taken from the neighborhood is apt to have a better color. Where subcutaneous tissue is also desired, as over the bearing surface of a bone that is covered only by scar, the pedicle graft is more appropriate. The final result of the pedicle flap is often greatly compromised by the excess of subcutaneous fat that is included. This is most noticeable with flaps transplanted to the hands or fingers. The question of the possible subsequent increase of fat in the transplanted flap concurrent with a later general increase of the fat has not, as far as known, been settled. The "Thiersch" graft is also more certain of taking, the technic of its use is less exacting and it can be applied within the mouth as well as on a clean skin surface. On the other hand its subsequent contraction is many times greater than a successful Wolfe graft and its color and appearance is not as good.

The full thickness graft is somewhat uncertain of result, is exacting in its technic, requires three weeks before it can be considered safe, and in children and young adults may develop red corded scars months after an apparently perfect take, yet it has a place in surgery that is not as well filled by either of the two preceding.

The Technic. The technic was as follows: The graft is usually implanted either in the bed from which a flap or a scar or growth has just been removed, or in the space that results where a tight area of skin or scar has been incised and released. The bleeders are caught and, where practicable, tied with fine silk, preferably white. A pattern accurately reproducing the shape and size of the raw surface is cut from a piece of sterile tin-foil and the side corresponding to the epithelial surface is indicated by writing on it with a blunt instrument the name and date. This has proven to be important. The skin from which the graft is to be cut is painted with 5 per cent picric acid in alcohol, which is later sponged off with alcohol. The tin-foil pattern is laid on the skin, epithelial surface up, and the outline very accurately marked by a knife cut which immediately

or later is made to go through the full thickness. Allis, or small Ochsner forceps grasp at intervals along the edge of the skin at the end of the outlined graft, while with a very sharp knife the skin is released from all subcutaneous vessels and tissues, cutting always where the junction of the skin with the subcutaneous tissue is most tense. The under surface of the skin should show white and stippled with little pits, and should show no bleeders. The graft is given in charge of an instrument nurse, who places it in a pan, folding it so that raw surface is to raw surface and the whole covered with damp gauze. When it is to be used one assistant grasps an edge with an Ochsner or Allis forceps and is responsible for it until the graft is safely tacked in place. This bit of formality may save the embarrassment of losing the graft among the discarded sponges or of dropping it on the floor.

The graft is usually punctured and then sutured into its new bed with a continuous horsehair, first tacking it in a few places to assure accuracy of position and even tension. If the vessels of this bed have not been tied, as may be the case in a burnt area, bleeders must be controlled between the removal of the forceps and the application of the pressure dressing by sponge pressure or an Esmarch bandage. Just before the final dressing is applied the clots are pressed from under it by firmly rolling over the surface a roll of gauze; the surface is then covered with several layers of gauze impregnated in 3 per cent xeroform ointment. Over this is applied a large, soft, damp marine sponge which is bandaged in place with some pressure. The quality of the sponge and the tension on the bandage are important factors.

NOTE.—We use a good quality of bath sponge which when dry is about $1\frac{1}{2}$ inches thick by 4 x 5 inches in diameter. These are washed, soaked in an antiseptic, rinsed, dried and put away in a jar. When they are to be used they are wet with saline and wrung out as dry as possible in a towel. Sponges may be cleansed and used repeatedly, but when they lose their elasticity they must be discarded.

If the vessels have not been tied and the bandage pressure is depended upon to control the bleeding, it is well to put an inner bandage over the sponge at the tension it is desired to maintain until the first redressing, and over this place a much tighter bandage that is to be removed in three or four hours. The sponge pressure of the permanent dressing should be firm, but not sufficient to cause ischemia of the graft. One's surgical sense may be severely taxed in figuring this out. Blood seeping into the sponge can make the pressure greater than it was when the bandage was applied. All gauze dressings should be laid on smoothly, the sponge should be soft and the gauze not wrinkled during the bandaging, as patterns of damaged epithelium have been observed that seemed to correspond to wrinkles in the gauze dressings.

The space that furnished the graft may be treated in one of several ways. At first we excised the fat, undermined the edges and obliterated it with sutures. This causes additional bleeding and delay, and should the sutures not hold, the scar will be fixed to the aponeurosis or muscles. Now, we simply approximate the borders with silkworm gut and either plicate the raw surface with the suture or undermine the edges by thrusting a dissecting scissors and opening them as they are withdrawn. This causes little bleeding. It is always safe to drain the wound at intervals. If the defect is too large to close, it is "Thiersch"- grafted from the thigh with or without encircling it with a purse-string suture.

If satisfied as to the asepsis, the original dressing can remain undisturbed for three weeks, but usually it is safer to look at a graft on the young child or infant in five or six days and the adult's in seven or nine days after operation. If the pressure dressing is for any reason removed before these dates it should be replaced in the same manner as at the original operation. Where mouth secretions or vomit are apt to soil the dressings, a dam composed of rolls of vaseline gauze may be plastered to the lip at the edge of the dressing. Leaving off the pressure dressing at too early a date favors the formation of blebs, which in turn predispose to ulceration of the epithelium. The base

of blebs that have or threaten to become infected are painted with 1 per cent silver nitrate once a day and a xeroform ointment dressing applied. It is not safe to use a dry dressing. The dressing problem is very much simpler if the child has not been hurt by pulling off dressings that have stuck. Of late, as already stated, a boric-acid pack has been used for the later dressings on infected cases, which seemed to clear off the slough and stop the infection quicker than did the silver and ointment. If a moist dressing is used it must be kept moist. Lost areas of epithelium can be replaced with a "Thiersch" graft put on under pressure as soon as the slough and infection have cleared up.

DISCUSSION

Dr. John Staige Davis, Baltimore, Md.:

I am very glad that Dr. Blair has read this paper on "Full Thickness Grafts." This type of skin graft is most satisfactory, and I have used it with success for many years.

The sea-sponge dressing for skin grafts, first used by Dr. Blair for this purpose, is wonderfully effective, and it is also very valuable for dressing other wounds, especially where even pressure is required. The excellent results shown on the screen should stimulate others to use whole thickness grafts more frequently.

Dr. Charles H. Mayo, Rochester, Minn.:

I have seen a number of these cases of Dr. Blair. The patients are picturesque to say the least. There are only a few men who have the particular artistic sense and the ability to carry on this work.

I am reminded here of an idea I have never been able to put across, and I am taking this opportunity to make another attempt. It was a case of a lost scalp. The skull was dry and hard, the bone showing over a considerable area. I drilled holes half an inch apart, and in ten days I had a "mushroom" coming up through every hole. That gave me an opportunity to graft and a field to graft on. The result was satisfactory, and I recommend the method.

Dr. Blair (in closing):

Dr. Mayo's plan of drilling into this bone might be compared to a well-functioning stomach; nobody ever talks about it, but it is always in use.

If we can find out what causes the tanning of these grafts, then we will have advanced a great step.

REPORT OF THE AMERICAN COLLEGE OF SURGEONS COMMITTEE ON THE TREATMENT OF MALIGNANT DISEASES WITH RADIUM AND *X*-RAY

By ROBERT B. GREENOUGH, M.D. (Chairman)
BOSTON, MASS.

FEBRUARY 1, 1924.

TO THE BOARD OF REGENTS, AMERICAN COLLEGE OF SURGEONS, PHILADELPHIA, PA.:

Your committee respectfully submits the following report: In February, 1922, this committee was appointed and at the present date—February, 1924—is composed of the following members:

C. F. BURNAM,	Baltimore
G. W. CRILE,	Cleveland
WILLIAM DUANE,	Boston
J. M. T. FINNEY,	Baltimore
BURTON J. LEE,	New York
H. A. PANCOAST,	Philadelphia
H. GIDEON WELLS,	Chicago
FRANCIS C. WOOD,	New York
R. B. GREENOUGH,	Boston (*Chairman*)

Chairmen of local subcommittees and associate members of the general committee have been appointed as follows:

J. M. T. FINNEY,	Baltimore
R. B. GREENOUGH,	Boston
HARVEY R. GAYLORD,	Buffalo
ALBERT J. OCHSNER,	Chicago
GEORGE W. CRILE,	Cleveland
C. JEFF MILLER,	New Orleans
CHARLES H. PECK,	New York
JOHN G. CLARK,	Philadelphia
CHARLES H. MAYO,	Rochester
ALEXANDER PRIMROSE,	Toronto

The result of treatment of cancer of the cervix of the uterus was selected as the first subject for investigation. Abstract cards were prepared by the committee and the coöperation of the established clinics in the various cities represented was sought and obtained. To the many individuals who have taken part in the investigation, and especially to those junior men who took on the laborious task of transferring the clinic records to the abstract cards, the thanks of the committee are due. Without their interest and coöperation the investigation would have been impossible.

The cases selected for study were all treated in the years 1914–1919 inclusive. Each clinic was requested to provide a record of the cases of cancer of the cervix which entered the hospital within that period, whether treated by operation, by radium or by any other method.

1210 case records have been collected by the committee for analysis. These records come from the following hospitals:

Free Hospital for Women, Boston	Dr. W. P. Graves
Huntington Hospital, Boston	Dr. G. A. Leland
Massachusetts General Hospital, Boston	Dr. Lincoln Davis
Augustana Hospital, Chicago	Dr. Henry Schmitz
Cook County Hospital, Chicago	Dr. Henry Schmitz
German-American Hospital, Chicago	Dr. Henry Schmitz
Post-Graduate Hospital, Chicago	Dr. Henry Schmitz
St. Mary's Hospital, Chicago	Dr. Henry Schmitz
St. Joseph's Hospital, Chicago	Dr. Henry Schmitz
Washington Boulevard Hospital, Chicago	Dr. Henry Schmitz
Frances Willard Hospital, Chicago	Dr. Henry Schmitz
Roosevelt Hospital, New York	Dr. Howard C. Taylor
University Hospital, Philadelphia	Dr. John G. Clark
Philadelphia General Hospital, Philadelphia	Dr. Charles P. Norris
Scranton Hospital, Scranton, Pa.	Dr. J. M. Wainwright
Presbyterian Hospital, New York	Dr. James A. Corscaden
Toronto General Hospital, Toronto	Dr. Alexander Primrose
Howard A. Kelly Hospital, Baltimore	Dr. Curtis F. Burnam
Jefferson Hospital, Philadelphia	Dr. B. A. Anspach
Woman's Hospital, New York	Dr. George Gray Ward
Memorial Hospital, New York	Dr. Wm. P. Healey
Lakeside Hospital, Cleveland	Dr. George W. Crile

It may be said that the results here recorded represent a considerable number of the clinics which are believed to be well equipped in every way to deal with this disease, either by radiotherapy or by radical operative measures.

The problem before the committee was to obtain data in regard to cases of cancer of the cervix treated by operation, by radium or *x*-rays, or by any other method, or combination of methods, of treatment. In order that a fair comparison of the results of treatment should be possible, it was necessary that the same rigid conditions should obtain in determining the results of all methods of treatment as have been agreed upon by the surgical world to be necessary in reporting the end-results of the operative treatment of cancer in its various situations. It was also necessary, especially when dealing with the cases treated by radiation, to obtain a classification of cases according to the extent of disease present at admission to the hospital, so that early favorable cases treated by operation should not be compared directly with advanced cases subjected to radiation only as a palliative measure. Such a classification of cases, based upon the clinical findings on admission and corrected when possible by subsequent operative findings, has been procured.

The correction of the clinical classification of cases by subsequent operative findings undoubtedly leads to a more correct estimate of the actual conditions; but it should be recognized that cases which are not subjected to operation do not obtain this correction, and this fact, therefore, works somewhat to the disadvantage of the cases treated by radium or other non-operative procedures.

Before presenting the statistical result of the investigation a few comments upon the conditions seem to the committee to be appropriate. In order to obtain a sufficient number of case records, and in order to have a sufficiently long period after operative treatment to estimate the end-results on the arbitrary three-year basis, it was necessary to set the period of investigation for the calendar years 1914, 1915, 1916, 1917 and 1918. In 1923 this was extended to include the year 1919.

During this period, which includes the years of the war, hospital services were somewhat disorganized, clinical work on these problems was interrupted, and hospital records were not always as fully kept up to date as could be desired. The radical operative treatment of cancer of the cervix had been fairly well standardized, and was in general use in operative clinics. Radium therapy, during these years, however, was in a state of constant experimental modification. The dosage employed was, as a rule, less than in these same clinics at the present time, 1924, and the application of radium was less accurately accomplished. Repeated treatments were employed in many cases in contrast to the single massive dose administered under anesthesia, and with all the conditions of a surgical operation, such as is the usual technic at the present time. It may be said that it is the general opinion of radiotherapists that the results of radium therapy during the period under discussion were far less favorable than are those to be anticipated from the technic now employed. Since the results of the present method, however, cannot be judged for at least three years to come, it is believed wise to consider what was then accomplished and to await the end-results of the present methods as they mature. We must admit, however, that, as between radium and radical operation, these conditions are to the disadvantage of radium in no small degree.

During the period concerned in this investigation, high voltage, short wave-length *x*-ray therapy was not in common use, and no cases were recorded in which *x*-ray was employed, except as an adjunct to other methods. To judge of the value of *x*-ray from these data is therefore quite impossible.

The standard surgical report of end-results in cancer cases, furthermore, demands pathological evidence of the presence of cancer. In operative cases this is readily obtained, as the tissue removed is submitted entire to the pathologist. In many cases suitable for radiotherapy, however, the removal of tissue for a biopsy cannot be accomplished without adding to the discomfort

of the patient or prolonging the operation, and for these reasons is occasionally omitted by the surgeon who feels himself quite competent to make a diagnosis on clinical evidence alone. Such cases, while in all probability actual cases of cancer, are of no value in this investigation, as they fail to fulfil the prescribed conditions, and thus must be omitted as inconclusive. There were 32 such cases in this series, 8 in which hysterectomy was followed by radium and 22 in which radium alone, or combined with cauterization, was employed. All of these cases are alive and well, and their omission on account of lack of pathological evidence of cancer lowers the percentage of "cures" by radium in comparison with hysterectomy. We believe that due weight should be given to these facts in estimating the value of the different methods of treatment.

Attention should be called to the use of the word "cure" in the following pages. The term is used as the most convenient one to express the fact that the case in question is reported free from evidence of disease at a period three years or more subsequent to treatment. It is not meant to imply that subsequent recurrence of the disease may not take place. It is the purpose of the committee to obtain later reports on all these cases.

While the total number of case records is already large, constant additions to this number are to be expected, and supplementary reports will be prepared from time to time and presented by the committee. When the total number of cases is divided into the different classes, however, and when these numbers are again subdivided into smaller groups, according to the method of treatment employed, the number of cases in each group are much diminished; to such an extent, indeed, in certain groups as to make possible serious error if too much weight be placed upon variations of slight degree in the percentages obtained. The committee does not wish to be accused of failing to recognize these facts, but draws attention to them that no misunderstanding of the conditions may occur. Such as they are, however, and pending the receipt of additional records, which may

tend to overcome this objection by providing a larger number of cases in each group, the results are here reported.[1]

TABLE I.—CANCER OF THE CERVIX

Total cards received		1210
Discards:		
Untraced	159	
Not cancer of cervix	10	
No treatment	56	
No pathological evidence	32	
Under three years	25	
Reëntries—Duplicates	23	
1913 and 1920, 1921, 1922	43	
No data to classify	13	
Otherwise inconclusive	20	
	—	381
Satisfactory for study		829

It was necessary to reject 381 records of the total number of 1210. The reasons for discarding these records are stated in the table.

[1] By the courtesy of Prof. E. B. Wilson, of the Harvard School of Public Health, the following explanatory footnote has been added to this report:

The mathematical formula commonly used by statisticians to indicate the influence of chance in determining percentage relations is expressed as "Standard Deviation." This term is meant to indicate that chance fluctuations would be expected to lie, at least two-thirds of the time, within the assigned limits. The formula is as follows: Let N represent the number of cases recorded, and F the fraction "cured." The standard deviation would be $\sqrt{F(1-F) \div N}$. Taking the figures from Table III, as applied to Class 1.A; $N = 123$; $F = 35/123$, or 0.28. The standard deviation would then be $\sqrt{.28 \times .72 \div 123}$. This is 0.04+. In terms of percentage this indicates that at least two-thirds of the time the chance fluctuations of the 28 per cent recorded in Table III would lie between 24 per cent and 32 per cent. The standard deviation has been calculated in this way for the percentages given in these tables, and is noted in the text in reference to those tables where it is a matter of significance.

Another formula, "Probable Error" is two-thirds of standard deviation, and indicates that one-half of the time the fluctuations due to chance would lie within the assigned figures. Standard deviation has been used in this paper in preference to probable error, as being the more rigorous test.

32 cases were rejected as they lacked pathological evidence of the presence of cancer; these cases were, however, all alive and well more than three years after treatment.

25 cases were rejected because less than three years had elapsed from the time of the last treatment; these cases also were alive and well.

5 cases, however, were accepted as three-year "cures," in which a period of three years had not elapsed after the last treatment with radium. These were all cases in which the original treatment was given more than three years before, and the later radium treatment was given, without evidence of recurrence, only as a prophylactic measure. 1 of these was a case of hysterectomy with postoperative prophylactic radiation, 1 cautery and radium, and 3 were treated by radium alone.

829 cases remain as satisfactory for study.

TABLE 2.—CANCER OF THE CERVIX

Primary cases		688
1.A—Early favorable	123	
1.B—Borderline	120	
1.C—Broad ligaments involved	310	
1.D—Advanced cases	135	
	688	
Recurrent after hysterectomy		123
Cancer of cervical stump		18
Total		829

688 primary cases were divided into four groups:

1.A—Early favorable,
1.B—Borderline.
1.C—Broad ligaments involved.
1.D—Advanced cases.

There were 123 cases of recurrence after hysterectomy, the hysterectomy in these cases having been done in some other institution. None of these cases appear in the 688 primary cases above recorded.

There were 18 cases in which cancer of the cervix developed in the stump left after a supravaginal hysterectomy for fibroid.

TABLE 3.—CANCER OF THE CERVIX

RESULTS

ALL METHODS OF TREATMENT

Primary cases:	Cases.	"Cures."	Per cent.
Class I.A	123	35	28.0
Class I.B	120	22	18.0
Class I.C	310	24	7.0
Class I.D	135	2	1.4
	688	83	8.3
Class 2 and 3 (recurrent after hysterectomy)	123	9	7.3
Class 4 (cervical stump)	18	2	11.0
Totals	829	94	11.0

Table 3 records the results of treatment in the different classes of cases. As would be expected, the greatest number, as well as the greatest percentage of "cure," occur in the early and favorable cases.

There were 9 "cures" of recurrence after hysterectomy and there were 2 "cures" in cancer of the cervical stump. 94 cases of the whole number, or 11 per cent, were "cured" of their disease. It should be repeated that in all of these tables the word "cure" is used advisedly to indicate only a period of three or more years elapsed without evidence of disease. It is the intention of the committee to obtain later reports on all these cases.

All of the cases of this series entered the hospital in the calendar years 1914 to 1919 inclusive.

Table 4 indicates the number of years that have elapsed in the "cured" primary cases. It is to be noted that cures of six or seven years' duration have been obtained by hysterectomy with

or without radium, and by radium, or radium with palliative operation.

TABLE 4.—CANCER OF THE CERVIX

PRIMARY CASES

"Cures."	Duration. 3 to 4 Yrs.	4 to 5 Yrs.	5 to 6 Yrs.	6 to 7 Yrs.	7 to 8 Yrs.	Total.
Hysterectomy alone	5	5	6	3	..	19
Hysterectomy and radium	1	1	..	4	2	8
Radium and hysterectomy	2	3	1	1	3	10
Radium alone	12	5	1	2	4	24
Radium and *x*-ray	1	1	1	1	..	4
Radium and cautery	2	6	5	..	3	16
Radium and amputation	..	1	..	..	..	1
Cautery and ligation	..	1	..	..	..	1
Total	23	23	14	11	12	83

TABLE 5.—CANCER OF THE CERVIX

829 CASES—94 "CURES"

	Cures.	Per cent.
By hysterectomy alone	19	2.2
By hysterectomy supplemented by radium	18	2.1
By radium alone or with *x*-ray	37	4.4
By radium and palliative operation	19	2.2
By cautery alone	0	
By cautery and ligation of internal iliacs	1	0.1
Total	94	11.0

Table 5 indicates the actual number of cases and the percentage of the total number which were "cured" by different methods of treatment. It will be seen that radium alone, or with *x*-rays, was responsible for 37 "cures," or 4.4 per cent of the total number, and radium and *x*-ray, with or without a palliative operation, gave 56 "cures," or 6.6 per cent of the total cases treated. This is to be taken merely as an indication of the absolute value of radium in the treatment of this series of cases of cancer of the cervix. It is not in any way an indication of the relative value of radium as compared to hysterectomy.

TABLE 6.—CANCER OF THE CERVIX

RESULTS OF TREATMENT (1)

688 PRIMARY CASES

	Cases.	Cures.	Per cent.
Hysterectomy alone	74	19	26
Hysterectomy with radium	70	18	26
Hysterectomy with *x*-ray	2	0	
146 hysterectomies.			
23 operative fatalities	..	..	16
Hysterectomy with preoperative radium	40	10	25
Hysterectomy with postoperative radium	30	8	27
Hysterectomy with or without radium	146	37	26
40 hysterectomies with preoperative radium.			
4 operative fatalities	...	..	10

TABLE 7.—CANCER OF THE CERVIX

RESULTS OF TREATMENT (2)—*Continued*

688 PRIMARY CASES

	Cases.	Cures.	Per cent.
Radium alone	363	24	6.0
Radium and *x*-ray	20	4	20.0
Radium and cautery	91	16	17.5
Radium with or without *x*-ray or palliative operation	478	45	9.0
Cautery alone	52		
Other methods	12	1	8.0
478 radium applications.			
1 operative fatality	...	..	0.2

Table 6 shows the results of different methods of treatment in 688 primary cases regardless of the extent of their disease. It is to be noted that the percentage of "cures" by hysterectomy alone (26 per cent) is the same as the "cures" in cases where preoperative or postoperative radium is employed. The operative mortality of 16 per cent following hysterectomy is also to be noted.

There were 146 hysterectomies performed in the 688 primary cases; 127 were panhysterectomies after the Wertheim technic; 9 were recorded merely as abdominal hysterectomies. In 6 cases only a supravaginal hysterectomy was done and in 4 cases vaginal hysterectomy was performed. It is a notable fact that 3 of the 4 vaginal hysterectomies resulted in cure.

52 cases were treated by cautery alone, and in 92 cases cautery was used in combination with radium or *x*-ray. In many of the hysterectomies cautery also was used as a preliminary measure, either prior to or as a first step in the radical operation. In 24 of the cautery operations the record stated that the Percy technic was followed. 5 of these cases appear in the list of cures, but in 4 of them the cauterization was supplemented by radium treatment, and in the other one a hysterectomy was done. No one of the 52 cases in which cautery alone was used resulted in cure.

Table 7 gives some further details of the results of treatment. It is to be noted that the preoperative use of radium in 40 cases, followed by hysterectomy, gave approximately the same percentage of "cures" (25 per cent) as when radium was used in postoperative treatment (27 per cent). It is to be noted also that the 40 cases of preoperative radiation did not appear to increase the hazard of operation materially, as only 4 of these cases died of operation (10 per cent).

The main purpose of this investigation may be said to have been to obtain a classification of cases with reference to the extent of the disease in order that a fair comparison should be possible of the results of different methods of treatment in similar cases.

Table 8 indicates the results of treatment in the I.A group of early favorable (operable) cases.

Hysterectomy without radium gave 14 "cures" in 41 cases, or 34 per cent. There were 8 operative fatalities, a mortality of 19 per cent and the use of radium either before or after operation did not appear to increase the percentage of "cures."

Radium alone gave 15 per cent, and radium combined with *x*-ray, cautery or other minor operative procedure gave 6 "cures," or 42 per cent.

TABLE 8.—CANCER OF THE CERVIX

RESULTS BY CLASSES

1.A—123 CASES

	Cases.	Cures.	Per cent.
Hysterectomy without radium	41	14	34
8 operative fatalities	..	..	19
Hysterectomy with radium	28	9	32
Preoperative radium	13	4	30
3 operative fatalities	..	..	23
Postoperative radium	15	5	33
No fatalities.			
Hysterectomy without or without radium	69	23	33
Radium alone	30	5	16
Radium and palliative operation and *x*-ray	14	6	42
Radium with or without palliative operation and *x*-ray	44	11	25

TABLE 9.—CANCER OF THE CERVIX

RESULTS BY CLASSES

1.B—120 CASES

	Cases.	Cures.	Per cent.
Hysterectomy without radium	16	4	25
6 operative fatalities	..	..	37
Hysterectomy with radium	15	5	33
Preoperative radium	6	3	50
1 operative fatality	..	..	16
Postoperative radium	9	2	22
No fatalities.			
Hysterectomy with or without radium	31	9	29
Radium alone	56	6	10
Radium with palliative operation	21	7	33
Radium with or without palliative operation	77	13	16

The numbers, however, are small and it is probable that we get a more accurate idea of the prospect of cure if we combine the cases treated with radium, either with or without the use of the cautery, getting 11 "cures" in 44 cases, or 25 per cent. Roughly stated, in early favorable cases hysterectomy gives 1 chance in 3 of cure, with an operative mortality of 1 in 5, where

radium, with or without the cautery, gives a chance of cure in 1 in 4 without mortality.

If to these percentages we apply the statistician's correction for standard deviation we find that the 34 per cent "cures" by hysterectomy should be recorded as ± 7.4 and the 25 per cent for radium (with or without cautery or *x*-ray) should be ± 6.5. Thus chance alone might bring about a variation for the hysterectomy percentage from 26.6 per cent to 41.4 per cent and for the radium cases from 18.5 to 31.5 per cent. It is readily seen that these bounds actually overlap and the difference in percentage is not of serious statistical significance.

The 1.B group represents the so-called borderline cases, those in which the disease has begun to involve the vaginal wall or the cavity of the uterus, but without extension into the broad ligaments.

Hysterectomy alone gave 25 per cent "cures" in 16 cases. The operative mortality ran as high as 31 per cent. Hysterectomy with radium gave 33 per cent "cures" in 15 cases. The cases in which radium was used before operation, while only 6 in number, gave 3 "cures," or 50 per cent.

Radium alone gave only 10 per cent of cures. Radium with palliative operation gave a showing equal to that of the hysterectomies with or without radium, and radium with or without a palliative operation gave 13 "cures," or 16 per cent.

It would appear that in the borderline cases the prospect of cure by hysterectomy is less; the use of radium before operation may possibly improve the outlook, and the operative mortality is higher than in the 1.A group.

Although the numbers are small, it would suggest that the use of radium supplemented by a palliative operation gave the greatest prospect of cure and without operative risk. The calculation of the standard deviation, however, indicates that the difference of percentage is not of great significance, viz, hysterectomy with or without radium, 29 per cent ± 8. Radium with palliative operation, 33 per cent ± 3.

TABLE 10.—CANCER OF THE CERVIX

RESULTS BY CLASSES

1.A AND 1.B (COMBINED)

	Cases.	Cures.	Per cent.
Hysterectomy without radium	57	18	31
14 operative fatalities	...	..	24
Hysterectomy with radium	43	14	32
Preoperative radium	19	7	36
4 operative fatalities	...	..	21
Postoperative radium	24	7	29
No fatalities.			
Hysterectomy with or without radium	100	32	32
Radium alone	86	11	12
Radium with palliative operation or *x*-ray	35	13	37
Radium with or without palliative operation	121	24	19

Table 10 combines the groups 1.A and 1.B, giving larger numbers to deal with. Roughly stated, hysterectomy without radium cures 1 in 3, with an operative fatality of 1 in 4.

Radium with a palliative operation cured about 1 in 3 and radium with or without palliative operation about 1 in 5.

If we calculate the standard deviation in these percentages we get: Hysterectomy with or without radium, 33 per cent ± 4.6; radium with or without palliative operation, 19 per cent ± 3.5.

Table 11 gives the results of treatment in the 1.C group where the disease was believed to have infiltrated the broad ligaments.

There was 1 "cure" and 2 operative fatalities in 13 cases treated by hysterectomy alone, or 7 per cent. The addition of preoperative or postoperative radiation increased somewhat the percentage of "cures," 16 per cent; the numbers, however, were rather small.

Radium alone cured 12 cases and radium with palliative operation 7 cases; the numbers treated, however, were large and the percentage small.

13 per cent were "cured" by radium and palliative operation and only 7 per cent in all the radium cases.

TABLE 11.—CANCER OF THE CERVIX

RESULTS BY CLASSES

I.C—311 CASES

	Cases.	Cures.	Per cent.
Hysterectomy without radium	13	1	7.0
2 operative fatalities	...	..	15.0
Hysterectomy with radium	25	4	16.0
Preoperative radium	19	3	16.0
No fatalities.			
Postoperative radium	6	1	16.0
No fatalities.			
Hysterectomy with or without radium	37	5	13.0
Radium alone	189	12	6.0
Fatalities	1	..	0.5
Radium and palliative operation or *x*-ray	52	7	13.0
Radium with or without palliative operation	241	19	7.0

TABLE 12.—CANCER OF THE CERVIX

RESULTS BY CLASSES

I.D—135 CASES

	Cases.	Cures.	Per cent.
Hysterectomy without radium	4	..	....
Hysterectomy with radium	2	..	....
Hysterectomy with or without radium	6	..	....
Radium alone	88	1	1.1
Radium with palliative operation	27	1	3.0
Radium with or without palliative operation	115	2	1.7

Table 12 shows the advanced and supposedly hopeless cases. 6 cases were operated upon, with or without radium, without success. 2 cases only appear in the three-year "cures"—one treated by radium alone and one by radium and cauterization. It is obvious that these 2 cases at least were erroneously classed as "hopeless." This is in all probability due to the difficulty in distinguishing fixation of the uterus, due to cancerous infiltration of the broad ligaments, from a similar infiltration of an inflammatory character.

TABLE 13.—CANCER OF THE CERVIX

AVERAGE DURATION OF LIFE—FAILURES

	Cases.	Months.
Hysterectomy alone:		
Class I.A	18	21
Class I.B	4	27
Class I.C	9	13
Class I.D.		
Radium alone:		
Class I.A	25	28
Class I.B	47	14
Class I.C	167	14
Class I.D	78	9
Radium, cautery, *x*-ray:		
Class I.A.		
Class I.B	12	9
Class I.C	40	13
Class I.D	22	9

Table 13 shows the duration of life in the cases which failed of cure. It will be seen that no material difference exists as between the different methods of treatment.

In the early favorable group radium alone gave a slightly longer average of life, but in the less favorable cases this is not so apparent.

4 cases in the I.B group treated by hysterectomy averaged twenty-seven months. These numbers, however, are so small that little reliance should be placed upon this isolated observation.

TABLE 14.—CANCER OF THE CERVIX

COMPLICATIONS: FISTULA—R-V AND V-V

688 PRIMARY CASES

	Cases.	Fistulæ.	Per cent.
Spontaneous	688	8	1.1
After hysterectomy alone	74	4	5.5
After cautery alone	52	3	5.6
After radium alone	363	21	5.7
After hysterectomy and radium	70	4	5.6
After cautery and radium	91	7	7.0
Other methods	38		
		47	

Rectovaginal and vesicovaginal fistulæ occur spontaneously in cases of cancer of the cervix. There were 8 such cases in the entire series of primary cases.

The danger of fistula formation is apparently increased by any method of treatment, thus hysterectomy alone gave 5.5 per cent; cautery alone, 5.6 per cent; radium alone, 5.7 per cent; cautery and radium, 7 per cent.

It would appear that the danger of fistula formation should not weigh heavily in the choice of methods of treatment.

TABLE 15.—CANCER OF THE CERVIX

LOCAL HEALING—688 PRIMARY CASES

	Cases.	Cures.	Local healing.
Hysterectomy alone	74	19	3
Hysterectomy and *x*-ray	2	0	1
Hysterectomy and radium	70	18	13
Radium alone	363	24	37
Radium and *x*-ray	20	4	6
Radium and palliative operation	95	17	14
Cautery alone	52	0	1
Other methods	12	1	0
		83	75

The value of radium as a palliative measure in the treatment of incurable cases is generally admitted. That life is materially prolonged by the use of radium is difficult to prove.

Local healing of the disease in the cervix or vagina was noted, however, in 75 cases which failed of cure. This, of course, means relief from the offensive discharge and hemorrhages which are characteristic of the advanced stages of the disease.

Table 15 indicates the methods of treatment which resulted in local cure. The small group of cases in which radium and *x*-ray were used is perhaps most striking. Of 20 cases 4 were "cured" of their disease and 6 obtained local healing. Thus in 50 per cent of this small group the local lesion was destroyed.

TABLE 16.—CANCER OF THE CERVIX

RESULTS BY METHODS OF TREATMENT

	Cases.	Cures.	Per cent.
Recurrent after hysterectomy	123	9	7
By radium alone	...	7	
By radium and cautery	...	2	
Cancer of cervical stump	18	2	11
By radium alone	...	2	

The cases which showed recurrence after hysterectomy gave 7 "cures" by radium alone and 2 "cures" by radium and cautery. Of 18 cases in which cancer of the cervix developed after a previous hysterectomy only 2 were "cured," and those by radium alone.

TABLE 17.—CANCER OF THE CERVIX

RADIUM DOSAGE (LOCAL)

MILLICURIE HOURS

	Cases.	Average mc.hr.
"Cured" primary cases	57	5004
1.A Class	16	3354
1.B Class	18	5896
1.C Class	23	5455

An estimate of radium dosage in millicurie or milligram hours alone is misleading. The amount of infiltration and the distance also must be considered. It is an interesting fact, however, that the average amount of local or contact radiation employed in the successful cases was 5004 millicurie hours, irrespective of deep gamma radiation or *x*-ray applied externally. Such an amount of radiation can be obtained only by very prolonged treatments—four to eight days—when radium tubes of only 25 to 50 mg. are used. The smallest dosage of radium used in the "cured" cases was as follows:

Class A	1700 mc.hr.
Class B	1725 mc.hr.
Class C	2200 mc.hr.

The largest dosage in a "cured" case was 16,000 millicurie hours given in twenty-nine treatments.

In general, during this period there has been a distinct tendency to abandon the many times repeated small dosage local treatment in favor of the single massive dose of radiation administered under an anesthetic and with all the conditions of a surgical operation. In certain well-equipped clinics this is further supplemented by heavy gamma radiation or *x*-ray.[1]

TABLE 18.—CANCER OF THE CERVIX

Varieties of Cancer

83 "cures"

	Cures.
Adenocarcinoma	8
Adenoacanthoma	1
Carcinoma	14
Epidermoid carcinoma	5
Squamous-cell carcinoma	42
Solid carcinoma	1
Cylindrical-cell carcinoma	1
Epithelioma	7
Papillary carcinoma	4
Total	83

Table 18 shows the different varieties of cancer in 83 "cured" cases. It is apparent that no uniform method of classification has obtained wide acceptance in the clinics here represented. It is of interest, however, to note that the milder forms of the disease—adenocarcinoma and papillary carcinoma—are not by

[1] At the suggestion of one of the members of the Committee the following statement is made to supplement the report:

Much variation is found in the technique employed in radium therapy in this series of cases; methods were constantly changing in the period covered; and it is probable that during this time methods have been materially improved. It is the purpose of the Committee to submit a later report dealing with the details of treatment with radium, but it must be remembered that nothing short of the very best of radium treatment, as well as the best of surgical skill, should be considered adequate in dealing with this disease.

any means the only ones to be cured, whatever agency is employed.

It is also of interest to note that of all the cases in which hysterectomy resulted in a "cure," in only 2 was evidence presented that the disease had extended beyond the uterus itself, and in these 2 cases the pelvic lymph nodes were not diseased, but cancer was found by microscopical examination in the parametrial tissue adjacent to the cervix.

Summary. 1. As a result of the study of the case records submitted to the committee certain conclusions may be drawn. We would again emphasize the fact that this is to be regarded only as a preliminary report, and that it is the purpose of the committee to collect further data and extend the time beyond the minimum period of three years, so that knowledge of the more remote end-results of these cases may be obtained. In the meantime the figures available to the committee are submitted and such conclusions drawn as seem to be justified by the results.

2. 829 cases of cancer of the cervix showed 94 cases free from disease three or more years after treatment. More than half of these "cures" were obtained by the use of radium and *x*-ray without radical operation. There were no "cures" by cautery alone.

3. In 243 cases of the 1.A and 1.B group (early favorable and borderline cases) hysterectomy alone cured 1 in 3, with an operative mortality of 1 in 4. Radium with palliative operation (cautery) cured about 1 in 3 and radium alone—or with palliative operation—about 1 in 5. Under these conditions it may be said that the choice between operation and radium in the treatment of early and favorable cases of cancer of the cervix is an open one. It is to be borne in mind that the results of treatment by radium used with the technic of the present day are not yet available, but that it is generally believed that they will be better than the figures here presented.

4. In more advanced cases (1.C and 1.D) the "cures" either by radiation or by hysterectomy were very few.

5. The duration of life in the unsuccessful early cases is somewhat greater after radium than with operation.

6. The formation of rectovaginal and vesicovaginal fistulæ occurred with nearly equal frequency with all methods of treatment.

7. Radium, with or without *x*-ray or palliative operation, was the most important agency in the destruction of local disease in cases which failed to obtain a "cure." The value of radium as a palliative measure in advanced cases is beyond dispute.

8. In the treatment of recurrent cases after hysterectomy and in cancer of the cervical stump, radium is to be preferred to other methods.

9. A sufficiently large dosage of radium is necessary to obtain destruction of the local lesion. The treatment of cancer of the cervix with inadequate amounts of radium should not be encouraged.

10. A uniform classification of the pathological varieties of cancer of the cervix is much to be desired.

DISCUSSION

Dr. George W. Crile, Cleveland, Ohio:

First of all I believe both the medical profession and the public are under very great obligations not only to this committee, but especially to Dr. Greenough, for this important report.

I wish to refer to only one or two points:

First: Dr. Greenough has used very fine discrimination and fairness in making his conclusions and in leaving open certain points that are not clearly decided.

Second: I wish to say one word as to the word "cure." It is of no use to spend time in defining exactly what that term means in each individual case. We must, perforce, take it for granted that the word "cure" represents the opinion that the patient is all right.

In our clinic we have decided on the basis of this report to give radium and deep *x*-ray therapy a trial in all cases of cancer of the cervix until evidence shows us that this procedure is wrong.

Dr. F. C. Wood, New York:

It seems to me that Dr. Greenough has accomplished a very remarkable result. The statistics have been analyzed coldly and frankly and the conclusions drawn must stand as they are read. It is an interesting and important phenomenon to see the introduction of advanced statistical methods into surgical literature.

I was pleased to hear Dr. Lee's statement about omitting decimals. We are all very fond of decimals, but they have no value at all; even the significant figures often have a very restricted value. The first thing we have to learn in the study of statistical results is that we must grant large errors in such work. These statistics of Dr. Greenough point the way for future classification. Anything which this Association can do to encourage the continuation of this work I think is of extreme importance. We need accurate and careful studies, not of small groups of cases but of large. What we want is the statistics of the different cities, such as New York, Chicago, St. Louis and so on. Radium may be applied better in San Francisco than in New York, or surgery may be better in one place than in another. So it may be wise to advise radium in one city and surgery in another. The immediate value of such a collection at the present time is relatively slight, because only three years have elapsed, and it is of the utmost importance that the work be continued for at least five years more, so as to get significant figures. Where we have only 30 or 40 cases to discuss the percentages are of no importance.

It is therefore to be hoped that this Association will help Dr. Greenough in carrying this work on, and also include other sites of cancer.

Dr. C. H. Mayo, Rochester, Minn.:

It is interesting to see how many patients with cancer are benefited by the use of all known methods of treatment. In cases of cancer we must also take no chances, but use all reputable agents, and then try to analyze their action.

Last December, at the meeting of the Southern Surgical Association, Dr. John G. Clark and Dr. Howard A. Kelly, who formerly operated so much for cancer of the uterus and allied organs, both reported that they had seen such wonderful results from the use of radium that they had almost stopped operating in such cases. Radium and the *x*-rays have certainly made great headway in the cure of this terrible disease.

Dr. Greenough (in closing):

I would like to thank Dr. Crile and the other gentlemen for what they have said in regard to my personal efforts in this matter, but I would like to disclaim any reason for accepting them. You all know that statistical work, as it is done in the Department of Health, is done by a machine, and I have tried as far as I could to imitate that machine in collecting, sorting and presenting this material.

LIGATURE OF THE COMMON ILIAC ARTERY

By JAMES E. THOMPSON, M.B.B.S., Lond. Univ., F.R.C.S., Eng.
GALVESTON, TEXAS

Ligature of the common iliac artery is a comparatively rare operation. On an average about 1 case yearly has been reported since the operation was first performed by William Gibson, of Baltimore, Md., in the year 1812. Up to the year 1912 the reported cases are accessible in three articles: One by Stephen Smith, published in the *American Journal of the Medical Sciences*, July, 1860; the second by Kümmel, in the *Archives für klinische Chirurgie*, 1884, and the third by Halsted, published in full in the *Johns Hopkins Hospital Bulletin* in July, 1912, and in an abridged form in the *Annals of Surgery* and the *Transactions of the American Surgical Association* for the same year.

Up to 1912 Halsted was able to find references to 92 cases of ligature of the common iliac artery, of which 32 were collected by Stephen Smith, 30 by Kümmel and 30 by himself. To these I have been able to add 5 published cases and 3 unpublished, of which 2 are from the clinic of the John Sealy Hospital and 1 from the Mayo Clinic, an abstract of which was kindly sent to me by Dr. John Pemberton.

The infrequency of this operation probably depends on two factors, viz.: (1) On the rarity of aneurysms arising from the common iliac, and (2) on the fact that direct wounds of the vessel are usually fatal within a few minutes from rapid loss of blood. Aneurysms occupying the common iliac trunk usually encroach on the point of origin of the artery from the aorta to such an extent as to render ligature impossible or very dangerous. Most surgeons have preferred to place a ligature around the abdominal aorta rather than run the risk of applying a ligature

around a dilated and diseased artery in close proximity to the aneurysmal sac. In Case II of our series the difficulties were so great that we only succeeded in placing a ligature on the artery by dint of great perseverance and at considerable risk of penetrating the aneurysmal sac.

INDICATIONS FOR THE OPERATION. Stephen Smith divided the indications into four groups, as follows:

1. For the arrest of hemorrhage.

2. For the cure of aneurysms.

3. For the cure of pulsating tumors other than aneurysms (malignant growths).

4. For the arrest of hemorrhage during operations for the removal of growths.

GROUP 1. Although the first ligature of the common iliac was performed by William Gibson to arrest hemorrhage, surgeons in civil practice will seldom see cases in which the operation can be attempted with even a remote chance of success. Wounds of the artery are usually attended by such profuse hemorrhage that death follows in a few minutes before surgical treatment is available. Smith reports 11 cases in this group, of which 10 died, 9 from hemorrhage (primary 5, secondary 4) and 1 from peritonitis. In Halsted's list there were no cases of ligature for direct wounds of this vessel. There were 7 cases of hemorrhage of diverse origin from the common iliac trunk and its branches and the femoral and profunda arteries, resulting from varying conditions, such as free hemorrhage during the removal of pelvic tumors, diffused aneurysms, erosion of an artery by an ulcerating bubo and secondary hemorrhage after wound of the profunda femoris vessels. In these 7 cases there were 5 deaths.

GROUP 2. This is the most important, because an overwhelming majority of cases fall in it. The surgical history of ligature of the common iliac artery is indissolubly linked with that of aneurysms of the external iliac and hypogastric branches. In Smith's series of 32 cases there were 15 ligatures for the cure of aneurysms; in Halsted's 30 cases there were 23.

Group 3. At the present time surgeons would hesitate to countenance the operation for such an indication. Sufficient proof is available to show that ligature of the main artery distributed to a tumor has only a transient effect and cannot be considered seriously as a curative measure.

Group 4. At the present time other alternatives of a less serious nature would be employed. Either the hypogastric artery would be ligatured, as is the custom in extensive removal of uterine cancers, or the common iliac trunk would be obliterated temporarily during the removal of the tumor by compression with arterial clamps.

Anatomy. The common iliac arteries are the terminal branches of the abdominal aorta, which divides into two trunks opposite the middle of the body of the fourth lumbar vertebra a little to the left of the midline. Each artery passes from this point downward and laterally to the level of the lumbosacral articulation, where it divides into its two terminal branches, viz., the hypogastric and the external iliac. The right artery is a little longer (2 inches) than the left ($1\frac{3}{4}$ inches). On the surface of the abdomen a line, drawn from a point a little below and to the left of the umbilicus to another on the inguinal ligament, midway between the anterior superior spine of the ilium and the symphysis pubis, will indicate the course of the common and the external iliac arteries. The upper third will correspond to the common, the lower third to the external iliac. Both right and left arteries lie under the peritoneum and are covered by coils of the small intestine. Communicating cords of the sympathetic between the aortic and hypogastric plexuses also lie in fromt of the vessels. The superior hemorrhoidal vessels cross over the front of the left artery. Each artery is crossed obliquely near its bifurcation by the ureter of the same side. The left artery rests on the bodies of the fourth and fifth lumbar vertebra and the intervertebral fibrocartilages, on the sympathetic trunk and the psoas major muscle. The right artery is separated from these structures, except at its distal end, where it lies directly on the psoas major, by the left common

iliac vein, the commencement of the vena cava and the right common iliac vein. The left artery lies on the medial border of the psoas and has its companion vein lying to its medial side. The relations of the right artery to its vein are peculiar. Distally the vein lies slightly internal and posterior to the artery. As the vein ascends it lies directly behind the middle of the artery, and joins the left common iliac vein behind the proximal end of the artery, to form the vena cava inferior.

Both common iliac arteries are easily accessible by the intraperitoneal route. The small intestines are lifted out of the pelvis and retracted upward and the sigmoid flexure and pelvic colon are retracted to the left. An incision is now made through the peritoneal lining of the posterior body wall, below the line of the cecum and ileum. This is carried carefully across to the left side as far as the line of the superior hemorrhoid vessels. Through this opening both common iliac arteries can be exposed by stripping the peritoneum upward and laterally from the subjacent vessels. Care must be taken of the ureters on both sides. On the left side the superior hemorrhoidal vessels occupy a position medial to that of the left ureter. They must be retracted laterally. The common iliac arteries are also accessible by the retroperitoneal route, which was employed almost exclusively by the older surgeons. If the abdominal wall is divided down to the retroperitoneal tissue by an incision beginning at a point about $1\frac{1}{2}$ inches above and internal to the anterior superior iliac spine and extending upwards almost to the tip of the twelfth rib, the peritoneum can be peeled medially from the posterior body wall far enough to expose the common iliac artery on either side and even to expose the abdominal aorta on the left side in a thin patient. The transperitoneal incision is to be preferred, because it enables the operator to determine the exact position and character of the pulsating tumor and gives easier access to the arteries. Further, if ligature of the arteries is impossible, it enables him to ligature the abdominal aorta with greater ease and safety.

Collateral Circulation. In order to understand the path by which blood reaches the arteries of the lower extremity after ligature of the common iliac artery, it is necessary to review the anatomy of its main branches, viz., the hypogastric and external iliac.

The *hypogastric*, in the fetus, represents the primitive artery of the hind limb, whereas the external iliac and femoral are later developments. The original artery of the leg was the inferior gluteal, which was continued downward as the popliteal and peroneal arteries. With the later development of the external iliac and femoral trunks the distal part of the inferior gluteal underwent atrophy.

The main trunk of the hypogastric artery is about $1\frac{1}{2}$ inches in length. It arises from the parent trunk at the level of the lumbosacral articulation and passes into the pelvis minor, where it ends opposite the upper border of the sciatic notch by dividing into two main trunks, an anterior and a posterior. From the posterior trunk three branches usually arise, all of which are distributed to the parietes. They are the iliolumbar, the lateral sacral and the superior gluteal. From the anterior trunk both parietal and visceral branches arise. The parietal branches are the obturator, inferior gluteal and internal pudendal; the visceral branches are the umbilical (superior vesical), the inferior vesical, the middle hemorrhoidal and the uterine. Of these parietal branches the iliolumbar and lateral sacral are by far the most important, because they anastomose freely with branches arising from the abdominal aorta, the former communicating freely with the lower aortic lumbar arteries and the latter with the medial sacral artery. After ligature of the common iliac trunk, arterial blood passes by means of these anastomoses into the hypogastric artery and thence distally into the lower limb by one or both of the following paths: (1) Up stream along the hypogastric and around the corner into the external iliac and so down into the femoral, or (2) down stream along the inferior gluteal (sciatic) through the crucial anastomosis and along the chain of perforating arteries into the lower end of the femoral or the popliteal.

The volume of blood passing along each of these channels will depend on the peripheral resistance. In cases of aneurysm of the external iliac artery, ligature of the common iliac is frequently followed by consolidation of the aneurysm. When this occurs, all the blood which flows into the main hypogastric trunk through the anastomotic channels between the aortic lumbar arteries and median sacral artery above and the iliolumbar and lateral sacral arteries below will be diverted distally along the inferior gluteal. If, on the other hand, the aneurysm does not consolidate, most of the blood will flow up stream along the hypogastric into the external iliac and very little will flow down the inferior gluteal.

The role of the obturator in these anastomoses must not be overlooked. In a case reported by Salomon a dissection of the body ten months after ligature of the common iliac showed that the obturator arteries were greatly enlarged.

There is no reason to believe that the visceral arteries (except perhaps the uterine) play any important part in the anastomosis. They are so small that the current of blood can hardly be expected to reach the main hypogastric trunk with sufficient pressure to overcome much resistance. In one of my cases ligature of the right hypogastric artery failed to influence pulsation in an aneurysm on the left side which had reappeared after ligature of the left common iliac artery.

The external iliac artery gives off two parietal branches at its distal end just above the inguinal ligament. One, the inferior epigastric, courses upward and medially behind the rectus abdominis muscle in its sheath and anastomoses above with the superior epigastric. The other, the deep circumflex iliac, courses laterally just above the lower border of the inguinal ligament, pierces the transversus abdominis a little beyond the anterior superior spine and courses around the iliac crest between the transversus abdominis and the internal oblique until it ends by anastomosing with the branches of the iliolumbar artery. Both these branches are of great value as anastomotic channels. They are of the utmost importance after ligature of the external iliac artery,

because they take most of the strain from the crucial anastomosis. They are scarcely of less importance after ligature of the common iliac. Therefore, care should be taken to plan the abdominal incision for ligature of the iliac arteries so that these vessels may escape injury. For this reason the rectus abdominis incision is not advisable. A median incision is preferable. If the retroperitoneal operation is contemplated the incision must not encroach medially on the line of the inferior epigastric artery.

From the above description it is evident that under normal anatomical conditions ligature of the common iliac artery is a reasonably safe operation, because the collateral channels are quite able to carry enough blood around the obstruction to keep the limb alive and in good condition. The dangers of gangrene are not great. It has been variously estimated by different writers from 15.6 per cent (Stephen Smith) to 33.3 per cent (Matas). Halsted takes exception to these figures as being much too high. He believes, after a careful analysis of the cases in which gangrene followed ligature, that it was mainly dependent on the condition present and was not produced solely by the ligature. He places the percentage at 3.3. In analyzing case reports, one finds that there is much greater danger of gangrene when the aneurysm occupies the path of the collateral channels than when it is situated on the main iliac trunks themselves. Thus aneurysm of the gluteal arteries, and iliofemoral aneurysms which extend as far as the origin of the profunda femoris, are more dangerous than aneurysms of the external iliac or those of the hypogastric proximal to its branches, and even those of the common iliac itself.

Halsted says that, "in a general way, the larger the artery or the nearer it is to the heart the less the impairment of circulation attending its ligation. The subclavian, for example, may be tied quite without fear of gangrene, whereas from ligation of the axillary artery the circulation of the extremity is somewhat endangered, but not so much as from ligation of the brachial. Peripheral gangrene has not been observed in consequence of ligation of the aorta. It may occur after ligation of the common

iliac, has occurred much more frequently after ligation of the external iliac and has followed ligation of the popliteal in a considerable percentage of cases." He quotes Kümmel, who writes, "It seems, naturally within certain limits, that the nearer the ligated vessel is to the central organ of the circulation the easier it is for the collateral routes, by means of the increased pressure from the heart, to develop. Astonishing as it may seem, it nevertheless appears to be a fact, as already stated, that the ligation of the common iliac less endangers the vitality of the lower extremity and makes easier the establishment of the collateral circulation than does ligation of a peripheral vessel, for example, the external iliac. I therefore do not hesitate, in the case of aneurysm of the external iliac and high femoral arteries, to express a preference for the ligation of the common iliac even when the ligation of the external iliac is possible, for thereby the definite cure of the aneurysm seems to be more certain of accomplishment and the safety of the limb less endangered."

One cannot subscribe unreservedly to all the above statements. It is more probable that the situation of the aneurysm has more to do with the incidence of gangrene than the site of ligature. It is difficult for us to believe that ligature of the external iliac artery is not a safer operation for iliofemoral aneurysms than ligature of the common iliac. The former operation leaves the hypogastric free to transmit blood directly into its peripheral branches by way of the crucial anastomosis, whereas the ligature of the common iliac forces the blood to pass through two sets of anastomoses before it reaches the femoral or popliteal trunks, viz.: (1) A proximal set between the aortic lumbar and medial sacral branches above and the iliolumbar and lateral sacral below, and (2) a distal set between the superior and inferior gluteal arteries above and the branches of the profunda femoris below (crucial). By the time the blood reaches the distal set (crucial anastomosis) the pressure must be very low indeed. The strain on these posterior anastomoses is not dangerous as long as the arterial anastomoses between the inferior epigastric and deep circumflex iliac and the superior epigastric and aortic

lumbar arteries are not interfered with. But if the iliofemoral aneurysm consolidates as the result of ligature, these trunks close up and all the strain is thrown on the crucial anastomosis with occasional disastrous results.

Further, we cannot accept the statement that ligature of the common iliac is preferable to ligature of the hypogastric for the cure of gluteal aneurysms. We believe that the hypogastric trunk should be tied whenever it is accessible and free from disease. It is $1\frac{1}{2}$ inches long, and if care is used there is no danger of injuring its companion vein or any of its parietal branches. After ligature the flow of blood into the lower limb down the external iliac is not interfered with and there is no danger of gangrene. We can, also, if conditions are favorable, expose the upper parietal trunks, viz., the iliolumbar and lateral sacral, and by preventing any blood reaching the gluteal arteries from above insure the consolidation of the aneurysm. If, on the other hand, the common iliac trunk is ligatured the circulation of the leg is much more embarrassed. Owing to the obstruction caused by the gluteal aneurysm lying right in the pathway of the crucial anastomosis, the chief burden of carrying blood into the thigh will fall on the inferior epigastric and deep circumflex iliac through their anastomoses with the superior epigastric and the aortic lumbar arteries above. It is true that a little blood will reach the hypogastric artery by way of the anastomoses between the iliolumbar and lateral sacral arteries below with the aortic lumbar and the medial sacral arteries above, and that this will pass around the corner up stream into the external iliac. Still both these streams are mere trickles compared to the full flow that would pass along the external iliac artery unhindered after ligature of the hypogastric artery only. We feel convinced, where ever it is possible, that the hypogastric artery should be ligatured for aneurysms of the gluteals, but that if the artery is diseased a ligature should be placed on the common iliac. After ligature of the common iliac trunk it is unwise to attack the gluteal aneurysm directly, because the dissection will invariably destroy important and essential collateral channels, and will probably precipitate gangrene.

I have been able to find histories of two cases of ligature of the common iliac artery in the records of the surgical clinic of the John Sealy Hospital. Both were performed for aneurysms, which occupied the distal portion of the common iliac trunk, its bifurcation and a large part of the external iliac artery. In both cases recurrent pulsation appeared a short time after the ligature, presumably due to the rapid opening of the anastomotic channels connected with the branches of the hypogastric of the same side.

CASE I.—*Spontaneous aneurysm involving the left common and external iliac arteries; ligature of the common iliac artery close to its origin; return of pulsation; attempted distal ligature of a large vessel entering the lower end of the sac a failure; enormous increase in size of the aneurysm; death.*

L. G. T., negro, male, aged fifty-four years, was admitted to the John Sealy Hospital August 4, 1915, suffering with an enormous pulsating tumor in the left iliac fossa, which was diagnosed as a spontaneous aneurysm of the common and external iliac arteries. It reached upward within three inches of the navel and downward almost as far as the inguinal ligament. The pulsation was vigorous and a loud bruit was heard over it. The femoral pulse was absent. Over the line of the femoral artery, about an inch below the inguinal ligament, there was an old, depressed scar which had been produced by a bullet about twenty years previously. There was no sign of pulsation in any of the arteries of the thigh or leg, but the circulation was fairly good. Both thigh and leg were edematous and the seat of pain. It was probable that the femoral artery had been severed by the bullet. We had no means of knowing what had happened to the femoral vein; at all events the collateral circulation, both arterial and venous, seemed to have been restored perfectly.

Previous Diseases. About twenty years ago he had contracted a sore on his penis, followed by a bubo in his left groin. About fifteen years ago he contracted gonorrhea. At the present time the Wassermann reaction is negative.

Operation. Under general anesthesia, August 16, 1915, the abdominal cavity was opened by a midline incision. The aneurysmal sac filled up the left iliac fossa, overlapped the brim of the pelvis and projected a considerable distance downward into the pelvis minor. The upper end of the sac projected upward so far that we were unable to isolate the left common iliac at the first attempt. The lower part of the abdominal aorta was cleaned and a piece of umbilical tape was carried around it. Before tying the ligature another attempt was made to isolate the common iliac artery. This was successful and we managed to clean about half an inch of the vessel, which seemed sound enough to hold a ligature. It was tied with umbilical tape. The abdominal aorta was not tied. Pulsation in the aneurysmal sac stopped at once. The patient made an excellent recovery from the operation. Several days after the operation the sac began to pulsate feebly. Day by day the pulsation got stronger, until at the end of two weeks, it was as vigorous as before the operation.

One morning I noticed that pressure over the lower pole of the sac, along the line of the femoral artery at the level of the inguinal ligament, lessened the pulsation. I found that by dipping my fingers deeply under the lower and inner edge of the sac and pressing firmly that pulsation ceased entirely. I felt certain that the femoral vessels had been destroyed by the bullet many years before, but I thought that perhaps an unusually large aberrant obturator artery was feeding the sac with blood. With this possibility in view, I operated on the patient again under local anesthesia. An incision was made along the line of the femoral artery. The dissection was carried deeply to the psoas muscle, but no signs of either femoral artery or vein were found. A careful search was made under the lower pole of the aneurysmal sac for the entering artery, but it ended in failure. Fearing lest we might inadvertantly open the sac the operation was abandoned. The patient refused to allow me to ligature the abdominal aorta. He was discharged from the hospital October 8, 1915.

He was readmitted March 7, 1916. At this time the sac was extremely large. It filled the whole lower abdomen and seemed to have extended across to the right iliac fossa. The upper pole extended above the umbilicus. Both pulsation and bruit were evident over the surface of the whole sac. The patient's general condition was fair. He suffered from intense pain in the left leg and thigh, in which there was considerable edema.

He refused to allow any operation to be performed after we had discussed candidly with him the dangers of ligature of the abdominal aorta. He was discharged March 26 and returned home.

I heard that he lived about six months.

Remarks. It was our belief at this time (1916) that the arterial blood, which kept the aneurysm alive and pulsating, reached the sac through the anastomoses between the visceral branches of the hypogastrics of the right and left sides, aided perhaps by those between the medial sacral and the lateral of the same side. If the pelvic cavity had not been completely filled by the aneurysmal sac I should have advised ligature of the hypogastric of the sound side, in the hope of cutting off these channels. In the light of our subsequent experience (in Case II) such an operation would probably have failed, because it is almost certain that the visceral anastomoses between the right and left hypogastrics are very feeble, even in the female (through the uterines), and that the vigorous anastomoses are homolateral between the upper parietal branches of the hypogastric and the lumbar branches of the abdominal aorta.

CASE II.—*Spontaneous aneurysm of the left external iliac artery, involving the bifurcation of the common trunk; ligature of the common iliac unsuccessful; subsequent ligature of the right internal iliac unsuccessful; distal ligature of the left femoral followed by immediate hemorrhage; old operation (Antyllus-Matas) performed at once, followed by a cure.*

C. F., colored man, aged twenty-seven years, was admitted to the John Sealy Hospital in June, 1922, suffering from bilateral

inguinal adenitis of gonorrheal origin. He had a clear history of syphilitic infection, and the blood Wasserman was 2 plus positive. The suppurating glands were removed from both groins. A few days afterward he complained of severe pain in the left groin, and we found a pulsating swelling along the line of the external iliac artery. This proved to be an aneurysm. September 2, through a transperitoneal incision through the left rectus muscle, the left common iliac was tied by my colleague, D. A. O. Singleton. Umbilical tape was used as the ligature. The left inferior epigastric artery was divided and tied while making the abdominal incision. The aneurysmal sac occupied the whole length of the external iliac artery and overlapped the bifurcation of the common trunk at its upper end. The patient was discharged at the end of a month, apparently cured. Convalescence had been ideal. The circulation of the thigh and leg was good and no pulsation could be detected in the sac. He was readmitted March 19, 1923, with a return of pulsation in the aneurysm. The sac was large and extended upward from the inguinal ligament below almost as high as the umbilicus above. It filled the whole of the left iliac fossa and extended to the right as far as the midline. Expansile pulsation was present. It was, however, less vigorous than before the operation. The bruit was well marked. The patient suffered from considerable pain, referred mainly to the terminal branches of femoral nerve in the thigh. March 28 the right internal iliac artery was tied with the braided silk. The abdominal cavity was opened by a median incision and the aneurysmal sac and left iliac arteries examined carefully. The sac had increased in size since the last operation. It now occupied the lower third of the common and all the external iliac trunks. Proximal to the upper end of the sac the common iliac trunk seemed to be obliterated. No pulsation was present over the line of the artery and we could find no trace of the ligature that had been placed on it six months before. The left internal iliac artery could not be found. It appeared to be covered by the upper end of the sac. The ureter was dissected off the lateral wall of the pelvis

and the side of the aneurysmal sac, and an unsuccessful search was made for the internal iliac artery and its branches. As a last resort, in the hope of cutting off the anastomosis between the branches of the right and left internal iliac vessels, the right internal iliac artery was ligatured. Pulsation was not very vigorous either before or during the operation. Compression of the right internal iliac appeared to stop it, and after the ligature was tied it was imperceptible. A few days after the operation pulsation and bruit returned, showing that we had failed to find the collateral arterial branch which was bringing blood into the sac. We were uncertain as to its location. Thinking that it might come from below through the branches of the medial and lateral circumflex arteries *via* the profunda femoris and femoral, we applied digital pressure on the femoral trunk just below the inguinal ligament. The test was unsatisfactory, because, on account of the scar tissue left by the operation on the suppurative bubo and the absence of pulsation in the femoral, it was impossible to tell if pressure had been applied correctly. It seemed more probable that blood was flowing into the sac through some of the branches of the internal iliac of the same side or perhaps through an aberrant obturator artery. The inferior epigastric and deep circumflex arteries were taken into consideration, but we did not think it likely these vessels could be responsible for the persistence of the aneurysm, because the lower end of the sac barely reached the level of the inguinal ligament. In addition, we had reason to believe that the inferior epigastric had been divided in the rectus abdominis sheath when the common iliac was tied. Believing that distal ligature of the femoral artery would stop the circulation through the sac, the operation was performed April 16, 1923. It was necessary to expose the artery thoroughly, in order to be sure of placing the ligature above the origin of the profunda femoris. To have shut off the profunda from the main femoral trunk would have been a blunder that would probably have been followed by gangrene of the leg, because it serves as one of the main collateral channels through which arterial blood reaches the thigh and leg after

ligature of the common or external iliac or common femoral trunks. The profunda arose at an unusually high level just below the inguinal ligament. The inguinal ligament was divided and the femoral artery followed upward until the lower end of the aneurysmal sac appeared. A silk ligature was placed on the artery about an inch below the sac. When it was tied it cut its way through the walls of the vessel as if they had been made of cheese. The proximal end of the artery began to bleed at once. It was seized with a pair of artery forceps, but the walls were so soft that the forceps would not hold. After several abortive attempts had been made to catch the bleeding end and tie it, the nose of the artery forceps slipped into the aneurysmal sac and a furious gush of arterial blood occurred. The opening was plugged with gauze while we held an anxious consultation over the case. We came to the conclusion that the old operation (Antyllus-Matas) offered the only good chance of success. It was necessary, however, to get control of the proximal circulation. We had already tied the common iliac and had nothing left but the abdominal aorta. Accordingly, we opened the abdominal cavity and exposed the abdominal aorta and passed a ligature around it. The ligature was not tied, but served as a loop against which the artery was compressed by the finger of an assistant. To our consternation compression of the abdominal aorta failed to stop pulsation in the sac. It was, however, impossible to abandon the patient, so we were under the necessity of opening the aneurysmal sac without control of the proximal circulation and trust to our dexterity and luck to reach and control the bleeding vessel. The sac was opened in front by a long incision which divided the abdominal muscles which covered it. Furious arterial bleeding came from the upper and hinder part of the sac, which was funnel shaped. The bleeding stopped when the funnel was blocked with the tip of the forefinger or when it was packed tightly with gauze. After trying in vain to get a view of the interior of the sac for the purpose of stitching up or clamping the bleeding aperture, we succeeded, with some difficulty, in carrying a strong ligature around the neck of the funnel

by means of a curved needle, which was passed from the interior of the sac, guided by the left forefinger, which served as a temporary plug. The ligature was tightened as the forefinger was withdrawn and knotted tightly. Our anxiety was intense. Happily, bleeding ceased at once. The rest of the sac wall was inspected, but no other arterial openings were found. The cavity of the sac was then packed lightly with iodoform gauze, the ligature withdrawn from around the abdominal aorta and the abdomen closed.

The patient made an uninterrupted recovery. The circulation in the leg was never embarrassed at any period. He left the hospital May 27, with a perfect leg. During convalescence he complained of pain in his left upper chest. *X*-ray examination showed a small aneurysm arising from the left side of the transverse portion of the thoracic aorta.

The most puzzling feature of the case was the failure to control circulation through the aneurysmal sac by compression of the abdominal aorta. It can only be explained by the opening up of the anastomotic channels between the lumbar branches of the abdominal aorta and the parietal branches of the internal iliac of the same side, as a result of the ligature of the common iliac seven months before.

Remarks. The return of pulsation after ligature of the common iliac artery was due to too rich an anastomosis. On the belief that the anastomotic channels were the branches of the right and left hypogastric arteries, we tied the right hypogastric artery (sound side) at the time of the second operation. It was my intention to have tied the left hypogastric artery (affected side), but it could not be found. The result was a failure, because our surmise as to the anastomotic vessels was incorrect. The final operation was planned as a distal ligature of the femoral artery, with the hope of arresting the circulation through the aneurysm, which occupied the whole length of the external iliac. If the ligature had not cut its way through the diseased vessel we might possibly have succeeded. But when the arterial wall gave way and bleeding started from the proximal end of

the vessel, events marched so rapidly that the tail wagged the dog for many anxious minutes. The aneurysm was opened and we had before us the task of arresting the hemorrhage. By good luck we took the right steps. If compression of the abdominal aorta had stopped the hemorrhage we should have been able to have stitched up the opening leading to the hypogastric artery without difficulty. As it turned out, we managed to suture it from within by a circular stitch. It is more than probable that if the abdominal aorta had been exposed and compressed at a higher level, *i. e.*, just below the renal arteries, that a bloodless field would have been secured.

Although the technic of the intrasaccular obliteration was somewhat atypical in this particular case, I believe that the operation can be considered as one of intrasaccular suture (Matas).

In a careful search through the literature of aneurysms of the iliac arteries I have been unable to find a similar case.

The following are abstracts of 5 published cases and of 1 unpublished:

Fee, F. Ligation of the Common Iliac Artery for Iliofemoral Aneurysms, with Report of a Personal Case. *Lancet-Clinic*, Cincinnati, 1915, cxiii, 594.

Case Report:—A man, aged forty-eight years, complained of pain in the right groin and leg. Symptoms were first noticed seven years ago and appeared the day after he was kicked in the groin, as a small, throbbing tumor (hematoma). The mass increased in size gradually and the pain became more severe until he was unable to follow his employment. For the past three years he has used crutches constantly. Examination showed a large, bulging tumor occupying the right lower quadrant of the abdomen. It reached vertically from the umbilicus to an inch below the inguinal ligament and laterally from the crest of the ilium to the midline. It showed all the physical signs of an aneurysm.

Operation: Transperitoneal route through a median incision about four inches long. A large pulsating mass occupying the right external iliac artery was found. The common iliac artery was tied with two stout silk ligatures placed about three-quarters of an inch apart. Pulsation in the aneurysm ceased immediately. The common iliac artery was not divided.

The patient made a very satisfactory recovery. Two years afterward examination showed entire absence of the aneurysm, no pulsation in the femoral; very slight pulsation in the posterior tibial and dorsalis pedis. There was slight capillary congestion of the affected leg, but no edema. There was almost complete restoration of function.

ARTH, O: Aneurysmus des Arteria iliaca und Vena communis dextra, *Arch. für. Klin. Chir.*, 1915, cvi, 823.

CASE REPORT: A male, aged twenty-two years, was wounded by a shot through the pelvis.

Symptoms: The right thigh was flexed on the abdomen; slight pulsation perceptible in the lower part of the right femoral artery; distinct resistant swelling above the inguinal ligament, but no pulsation. In Scarpa's triangle there was a pulsating swelling the size of an apple; great pain in the groin and upper part of the thigh.

Operation: Incision along the line of the external iliac artery; ligation of the inferior epigastric artery and vein; exposure of the external iliac artery vein around which a traction thread was passed, but not tied. Momberg's hemostasis was used to a limited extent and the aneurysmal sac was incised and the clot removed. In spite of considerable bleeding the common iliac artery and vein were exposed and the wounds in their walls isolated. Both artery and vein were ligated proximal and distal to the injury. The patient recovered slowly. There was no gangrene. He suffered considerably from cramps in the muscles of the leg and thigh. He died four months later of pulmonary tuberculosis.

PEMBERTON, JOHN DE J. (Mayo Clinic), 1919: Private communication, hitherto unpublished.

Man, aged forty-eight years. Left thigh amputated fourteen years ago for an aneurysm resulting from a gunshot wound received twenty-eight years ago. When examined, showed a pulsating tumor along the line of the femoral artery just below Poupart's ligament. Systolic pressure, 145; diastolic, 75; urine normal; Wassermann negative.

Diagnosis: Traumatic aneurysm of the left femoral artery, with extension along the external iliac as a fusiform aneurysm.

Operation: September 12, 1919. Ligature of the left common iliac artery a short distance from the aorta. Simultaneous ligature of the external iliac. Both common and external iliac trunks were greatly distended. Silk ligatures were tied around a segment of artery, which was first crushed with curved clamps.

Convalescence was uneventful. The patient was able to wear an artificial limb on the stump, without trouble most of the time, for four years after the operation. In August, 1923, he was seized suddenly with sharp pain in the stump, which was followed by swelling and soreness. *X*-ray showed slight roughening of the bone and a dense, calcified tumor in the soft tissues, which was interpreted as a calcification of the aneurysmal sac. September 22, 1923, the tumor was explored and a large sacculated aneurysm situated on the femoral artery below the origin of the profunda femoris was excised. The femoral artery was exposed above and below the origin of the profunda femoris and doubly ligated above and below this branch. The artery was hugely enlarged and measured $3\frac{1}{2}$ cm. in diameter. The excised aneurysm was filled with an organized laminated clot. A few calcareous plaques were found on the inner wall of the aneurysmal sac.

PRIEST: A Case of Ligation of the Common Iliac Artery for Femoral Aneurysm, *Jour. Missouri Med. Assn.*, St. Louis, 1921, xviii, 463.

Woman, middle-aged, first noticed a painless swelling the size of a marble in the right groin six years before admission. It

increased in size gradually, became painful and began to pulsate. Examination showed an expansile tumor with all the signs of an aneurysm along the line of the external iliac and upper part of the femoral arteries. The thigh and leg were quite edematous.

Operation: Transperitoneal incision; ligature of the right common iliac artery, which was greatly dilated, with braided silk. Immediate cessation of pulsation in the aneurysm, but return of pulsation, however, before the patient left the operating-table. Pulsation was still present, though not so vigorous as before the operation, when the patient was discharged on the fourteenth day.

Brooks, B: Aneurysm of the External Iliac and Femoral Artery. *Surg. Clin. of North America*, Philadelphia, 1922, p. 1657.

Case Report: Woman, aged forty-four years, complained of a swelling in the right groin which had been present six years. On examination an aneurysm was found occupying the right external iliac artery and the upper portion of the femoral. The pulsating tumor occupied the whole length of the femoral triangle. It had all the intrinsic and extrinsic symptoms of an aneurysm and was producing great pressure on the veins and nerves, causing edema and neuralgia.

Operation: Transperitoneal approach followed by ligature of the right common iliac artery in the middle of its length. Pulsation ceased at once, but reappeared slightly before the patient left the operating-table. During convalescence pulsation became more marked, but not to the preoperative intensity. The patient was seen thirteen months afterward, and one could see and feel pulsation, but the tumor did not appear to have increased materially in size. Pain was absent and edema had disappeared.

Eloesser, L: Aneurysm of the Common Iliac Artery: Gradual Occlusion by Ligation, with Free Graft of Muscle, *Surg. Clin. of North America*,, Philadelphia, 1923, iii, 681.

CASE REPORT: Male, aged sixty years, with a history of chronic fibroid pulmonary tuberculosis. Four years ago suffered from an abdominal crisis of doubtful nature. Two months later the left leg began to swell without fever or pain. This disappeared spontaneously. He suffered from several recurrent attacks, associated with swelling of both legs and undue frequency of urination. On examination the lower abdominal cavity was found filled with a huge pulsating expansile utmor. A diagnosis of aneurysm of the left iliac arteries was made.

Operation: Extraperitoneal approach and exposure of the left iliac vessels, which were found unconnected with the aneurysmal sac. The peritoneum was opened and the tumor palpated. It filled the whole pelvis and extended upward toward the right lumbar region. Here a large artery was felt, which when compressed stopped pulsation of the whole mass. Through a second retroperitoneal incision on the right side the pulsating tumor was exposed and followed upward as far as the lower pole of the right kidney, where the large arterial pedicle could be palpated. Little by little the index finger was hooked around it and two loops of stout catgut passed around the artery. A musculotendinous free graft was cut from the external oblique muscle, drawn around the artery and its free end stitched with catgut, so as to form a tight sling around the vessel. It was fastened tight enough to stop pulsation and to produce a thrill in the sac, whereas previously no thrill had been present. Pulsation reappeared soon after the patient left the operating-table, but it disappeared gradually from all the arteries of the right leg except the upper part of the femoral. There was a complete absence of thrill, bruit or murmur in the pulsating tumor. At the time of writing the tumor had steadily decreased in size. Pulsation was present in the right femoral as low as the origin of the profunda. Below this level there was entire absence of pulsation in any of the arteries of the leg. Both the legs were still edematous.

THE SURGICAL TREATMENT OF HEPATIC CIRRHOSES

By WILLIAM J. MAYO, M.D.
ROCHESTER, MINN.

Mann, in his classical experiments in the laboratories of the Mayo Foundation, removes the entire liver from dogs, and when they are about to expire a few hours later, he introduces a solution of glucose into their veins, which resuscitates them immediately; the period of resuscitation may be extended for from twenty-four to thirty-six hours by continuing the injection of the glucose solution at intervals, but all urea formation stops. This discovery emphasizes the metabolic functions of the liver, such as the metabolism of carbohydrates producing sugar for the heat and energy of the body. In the metabolism of proteins, also, the final steps in the development of the amino-acids take place in the liver, and in the liver, likewise, the fats are made fit for use in the body.

A function of the liver deserving of attention is that of the destruction of protozoa and bacteria that are removed from the blood stream by the spleen and intestinal organs of the portal system. Adami demonstrated that the sterility of the upper intestinal tract is due not only to the action of the gastro-intestinal secretions, but to some extent to phagocytes which pass out of the portal capillaries to the intestinal surface, pick up particles of fat and bacilli, and carry them into the radicles of the portal vein. The pigments of these microörganisms form the dark spots so common in the substances of the liver. Various bacterial toxins, and chemical poisons, such as arsenic and phosphorus, are to a great extent detoxicated in the liver.

The bile might be regarded as a by-product of the metabolic

processes in the liver, since the bilirubin of the bile is derived from the deteriorated red blood cells destroyed in the liver and elsewhere, and excreted as bile pigment, and the cholesterol content of the bile, a lipoid stored in fat, is liberated in the liver. Bile functions in intestinal digestion, especially in relation to the metabolism of fats, and many of the elements of bile, including water, are reabsorbed in the intestinal tract.

An interesting fact in connection with the function of the liver is that the liver acts only on non-oxygenated blood. Rowntree and Chaney are now carrying on in the Clinic certain interesting experiments in which arterial blood or oxygen is transferred to the portal circulation, to determine the direct effects of oxygen on the function of the liver.

The liver is peculiar in that all its cells are alike; consequently its diseases are of a simple pattern, as contrasted with those of organs with highly differentiated cells which introduce varied architectural possibilities. The star-shaped cell of Kupffer is not a true liver cell, but probably an endothelial cell of phagocytic type developed in the liver tissue spaces with specialized functions of a problematic nature.

The liver has remarkable powers of regeneration. Mann has shown that if 70 per cent of the liver of a dog is removed, it will be replaced within twelve weeks, not alone by hypertrophy, but by hyperplasia of the remaining liver cells.

When the liver is overwhelmed by an acute destructive poison, bacterial or chemical, its cells undergo acute fatty degeneration, regardless of the nature of the toxic substance which produces it, and in forty-eight hours the greater part may be converted into fat. When the toxic invasion is chronic, connective tissue is developed instead of fatty degeneration. The type of cirrhosis is determined by the route by which the toxic substances enter the liver; if by way of the portal circulation, portal cirrhosis results; if through the bile channels, biliary cirrhosis. In the use of the term cirrhosis I have been guided more by the clinical aspects of cirrhosis of the liver as a whole than by the etiologic factors or the minute pathologic histology.

In portal cirrhosis, the most common type of chirrhosis, failure to eliminate or detoxicate toxic substances carried to the liver by the portal system, leads to diffuse deposits of connective tissue around the portal vessels, which interfere with the hepatic circulation. Ascites, hemorrhage from the mucous surfaces, especially from the stomach, and other evidences of portal circulatory obstruction are the end-results of the vascular interference. The blood-pressure in the portal vein is about 30 mm., in the general circulation about 130 mm., and the back pressure on the portal circulation in portal cirrhosis is undoubtedly increased by the arterial counterpressure. The work of Segall, showing the effect of ligation of the various branches of the hepatic artery on the circulation of the liver, is most interesting in this connection. In Laennec's type of portal cirrhosis the liver is small, contracted, and nodular, but it should be noted that the cirrhotic liver may be normal in size, or considerably enlarged, and occasionally may be smooth from deposits of fat in the liver spaces.

There has been a tendency among pathologists, because of the varying morphology of the liver in portal cirrhosis, to describe each picture as a different type of cirrhosis, just as one might describe each pattern of wall paper or carpet as a different paper or carpet.

Fagge, of Guy's Hospital, London, early reported instances in which postmortem examination revealed very advanced portal cirrhosis in men who, apparently in perfect health, had died suddenly as a result of accidents. With characteristic sagacity, Fagge pointed out that through collateral vascular connections, especially those described by Sappey, the portal circulation had been reduced in these cases to a point at which the decompensated liver was able to care for the circulation without the development of serious obstruction, ascites or hemorrhage. Talma was the first to suggest the artificial establishment of a collateral circulation. Drummond, the physician, and Morison, the noted surgeon, working jointly on the same theory, introduced the operation of omentopexy to increase the collateral circulation.

and attempted to produce vascular adhesions between the surface of the liver and the abdominal wall.

The surgical treatment of portal cirrhosis by the Talma-Morison operation has given some good results. Of 47 patients operated on in the Clinic, 7 died in the hospital, 21 were alive when last heard from, 1 being alive and well more than nine years after operation, 1 eight years, 1 more than seven, and 1 more than five years. The operation we have usually performed is made through an epigastric incision, as for gall-stone disease, just to the right of the median line, which permits examination of the liver, and a second lower incision through the skin and muscle down to the posterior aponeurosis and peritoneum. The rectus muscle is separated from its posterior attachments and the omentum is drawn from above down into the extraperitoneal pocket thus formed. The extent of collateral circulation established in this way is extraordinary. In several instances I made an incision near by for other purposes, some time after omentopexy, and encountered so much venous bleeding that I had to desist. The Talma-Morison operation, *per se*, carries only a slight risk, but ascites, hemorrhages and often edema of the lower extremities of the patients for whom it is indicated make any operation extremely grave. The high mortality of the earlier Talma-Morison operations was undoubtedly largely due to the abdominal drainage through which peritoneal infections were subsequently carried. We have not used drainage, but have aspirated, whenever necessary, during the weeks succeeding operation, while collateral circulation is being established. The peritoneum in such patients is exceedingly vulnerable. Fagge demonstrated that 10 per cent of the patients who came to post-mortem through cirrhosis of the liver also had a terminal tuberculous peritonitis.

My interest in splenectomy for the relief of portal cirrhosis was excited many years ago by the remarkable benefit, such as the disappearance of ascites, hemorrhages, and other evidences of portal cirrhosis, which so often followed removal of the enlarged spleen in the Banti stage of splenic anemia. It is a fascinating

theory that in cases of splenic anemia the spleen, which belongs to the reticuloendothelial system, is enlarged primarily and the liver contracted secondarily, suggesting that the toxic substances are carried to the liver from the spleen, a splenic type of portal cirrhosis. This theory would also argue that in the common type of portal cirrhosis of the liver the liver is contracted primarily and the spleen enlarged secondarily. In other words, the toxic substances are not carried to the liver through the splenic portion of the portal vein, but through the gastro-intestinal portion of the portal vein, a true gastro-intestinal type of portal cirrhosis, and this is borne out by the fact that the enlargement of the spleen which occurs with the gastro-intestinal type of portal cirrhosis is not nearly so great as that with the splenic anemia type and secondary portal cirrhosis. This contrast is well shown in the gastro-intestinal type of portal cirrhosis of pepper and alcohol habitués, which is usually of the Laennec type, with only moderate enlargement of the spleen.

Experience in removal of the spleen in splenomegalilas of the splenic anemia type with secondary cirrhosis of the liver, encouraged removal of the spleen in cases of the gastro-intestinal type of portal cirrhosis of the liver. Forty-two of the 97 patients splenectomized for splenic anemia in the Clinic had more or less portal cirrhosis. Four patients died in the hospital, 21 are alive, 2 over nine years, 3 over seven years, 4 more than five years, and so forth. The results from splenectomy were extremely good, and contrasted with those from splenectomy in the gastro-intestinal type of cirrhosis of the liver, were remarkable, which again argues for an entirely different interrelationship between the spleen and the liver in these two conditions.

In 10 cases of splenectomy, with 3 deaths in the hospital, for advanced gastro-intestinal portal cirrhosis the results on the whole were disappointing; while the spleen was only moderately enlarged, it showed generalized thrombophlebitis and atrophy of the pulp cells comparable with that found in the spleens in cases of splenic anemia, and, considering the difference in the anatomic structure of the spleen, with the cirrhotic process found

in the liver. In 9 other cases splenectomy was combined with a Talma-Morison operation. Two of the patients died in the hospital. These patients were bad surgical risks and were operated on in the terminal stages; the results were none too encouraging as contrasted with the brilliant results following splenectomy for splenic anemia with secondary portal cirrhosis. The normal spleen furnishes about 20 per cent of the portal blood, the enlarged spleen much more. Splenectomy must necessarily reduce hepatic circulation. Perhaps part of the improvement may be due to the increased opportunity for collateral circulation in the vascular adhesions which form in the bed from which the spleen has been removed. I had occasion to operate for gallstone disease on a physician on whom twelve years previously C. H. Mayo had performed splenectomy for advanced splenic anemia with marked cirrhosis of the liver. It was with the utmost difficulty that I worked my way down to the encapsulated liver, which fairly floated in a venous plexus. The patient otherwise was in splendid condition. Removal of the spleen for these terminal conditions of primary cirrhosis of the liver entails considerable risk and should not be lightly undertaken, but in selected cases combined splenectomy and omentopexy should be considered.

What has been called "hepatic shock" is sometimes noted after comparatively slight operations on patients with very advanced decompensation of the liver, and in cases of portal cirrhosis the diagnosis will often be delayed or the operation will not be considered until the function of the liver is reduced to the point at which recovery is doubtful. There have been several unfortunate experiences of this kind in the Clinic.

Rowntree, on the medical service, Walters on the surgical service, and Greene, on the laboratory service of the Clinic, in collaboration have developed interesting and valuable facts from tests of liver function which demonstrate that, when the function of the liver is reduced to below 25 per cent, any serious operation will probably end fatally. Mann has shown that if the

function of the liver in the dog is reduced to a point below 20 per cent the animal will probably die.

In cases of biliary cirrhosis the liver is enlarged, dark colored, often soft, and has a tendency to bleed easily on slight injury. Biliary cirrhosis is most often the secondary result of infections and obstructive processes originating in the gall-bladder or the common duct, usually from gallstone disease. As Adami has pointed out, stone in the common duct, or obstruction in the head of the pancreas, is the usual cause of dilatation of the fine biliary ducts. The slowing of the circulation of the bile invites infections in and around the small biliary channels, and the resultant introduction of connective tissue around the minute bile ducts produces obstructions in the liver which lead to early and continuous jaundice. The spleen is not greatly enlarged in this type of biliary cirrhosis. The biliary tract should be cleared of obstructions, such as gallstones, and free drainage of bile established; the results are usually good. Splenectomy is unnecessary.

There is another type of biliary cirrhosis in which there is no demonstrable infection or obstruction in the biliary ducts. The liver is enlarged and congested, but firmer in consistency than in obstructive biliary cirrhosis. All the biliary ducts are greatly thickened and chronic jaundice exists. The spleen is enlarged and to a much greater extent than occurs in the obstructive type. It is interesting to speculate why the spleen is greatly enlarged in certain cases of biliary cirrhosis and not in others, and whether or not the splenic involvement is of definite significance. Hanot's name has been associated with an obscure type of hypertrophic biliary cirrhosis. I do not know just why his vague description, which has only added to the fog which surrounds the subject, should have resulted in an eponym.

Cases of what might be called the splenic type of biliary cirrhosis sometimes are graded according to the size of the spleen, but so far as I am able to judge clinically, the conditions associated with size are variations of essentially the same process. There are usually no gallstones nor infections in this group, but if they exist, they are apparently incidental. The disease is very

chronic and does not often present the acute symptoms exhibited in the obstructive type. Removal of the spleen sometimes seems to be indicated in these cases.

Just how removal of an enlarged spleen can be of benefit in such cases is a matter of speculation. The portal circulation in the liver is, of course, greatly reduced by splenectomy, and possibly this reduction is sufficient to reduce the amount of bile formed to the point where the obstructed channels can function. Splenectomy for hemolytic icterus has been extraordinarily successful, yet we know little concerning the disease. Perhaps a closer relationship between hemolytic icterus and certain splenic types of biliary cirrhosis may exist than is apparent on the surface. In 12 cases of splenectomy for the splenic type of biliary cirrhosis there was 1 death in the hospital; 5 patients are alive. It is evident that such patients, at present, come to operation in a terminal condition and are usually beyond the stage at which function of the liver can be restored.

There is a remarkable parallelism in the relation of an enlarged spleen to both portal and biliary cirrhosis. In the ordinary type of portal cirrhosis it is often possible to trace the origin of the disease directly to the gastro-intestinal tract (for instance, to the irritation of alcohol and pepper), and the spleen is not greatly enlarged; the results of splenectomy are only fair. The operations usually performed have been at a terminal period, too late. In terminal portal cirrhosis, seen in the Banti stage of splenic anemia, the spleen is very large, and splenectomy may be curative. In biliary cirrhosis of the obstructive type of Adami, due to obstruction and infections, such as are produced by gallstone disease, the spleen is not greatly enlarged, and there is no reason for splenectomy. Removal of the obstruction is sufficient. In these types of biliary cirrhosis in which there is no apparent infection or obstruction, in which the spleen is greatly enlarged, splenectomy may have value if not too long delayed. These facts lead to the tentative conclusion that there is a direct relation between the spleen and certain types of portal and biliary cirrhosis.

Summary. In the present incomplete state of our knowledge, cirrhosis of the liver may be divided into two fairly definite groups: (1) portal cirrhosis, the result of deposits of connective tissue around the radicles of the portal vein, causing ascites and hemorrhages from the stomach, and (2) biliary cirrhosis, the result of deposits of connective tissue around the biliary duct system, causing chronic jaundice.

The portal cirrhoses may be of two distinct types clinically: A primary gastro-intestinal type, sometimes definitely the result of alcohol, pepper or other irritating substances taken with food, in which the spleen is not greatly enlarged. In suitable cases, splenectomy and the Talma-Morison operation combined may have value. Splenectomy, in addition to the Talma-Morison operation, carries an increased risk, however, and before splenectomy is performed the functional capacity of the liver should be tested. In the secondary splenic type of portal cirrhosis, occurring in the late stage of splenic anemia, splenectomy gives splendid results.

The common forms of biliary cirrhosis are the results of obstructions and infections of the biliary ducts, usually associated with gallstone disease, and removal of these infections and obstructions, in cases not too far advanced, can be expected to result in cure. The spleen is not greatly enlarged in such cases and splenectomy appears unnecessary. There is a splenic type of biliary cirrhosis, however, very chronic in character, in which the splenomegalia is a prominent symptom, and splenectomy may be indicated.

THE FRACTURE COMMITTEE OF THE AMERICAN COLLEGE OF SURGEONS[1]

By C. L. SCUDDER, M.D.
BOSTON, MASS.

The Fracture Committee was appointed at the Washington meeting, May, 1922, of the American College of Surgeons. In view of the increasing importance of the subject of traumatic surgery, technically and industrially, and because of the very great interest of the surgeons of the American Surgical Association in the subject of fracture treatment, it seemed appropriate to present the status and accomplishments of this Committee on Fractures of the American College of Surgeons to this Association.

This Committee of the American College of Surgeons, having among its membership three members of the Fracture Committee of the American Surgical Association, which made its final report in 1921, may be said to have taken up the work of that committee and is trying to carry it a step forward. Those who have not read the volume containing the reports of the Committee on Fractures of the American Surgical Association published by the Association in 1922 will be repaid by its perusal.

The present committee is made up of eighteen men from all over the United States and Canada, who are specially concerned with fracture problems. The personnel of this committee is as follows:

[1] Read by title.

Nathaniel Allison,	Boston, formerly of St. Louis.
Astley P. C. Ashhurst,	Philadelphia.
Joseph A. Blake,	New York.
Fred. J. Cotton,	Boston.
William Darrach,	New York.
William L. Estes,	Bethlehem, Pa.
William E. Gallie,	Toronto, Canada.
Fraser B. Gurd,	Montreal, Canada.
George W. Hawley,	Bridgeport, Conn.
A. F. Jonas,	Omaha, Neb.
Paul L. Magnuson,	Chicago, Ill.
Lloyd Nolan,	Birmingham.
Robert B. Osgood,	Boston.
Charles L. Scudder, *Chairman*,	Boston.
William O'Neil Sherman,	Pittsburgh, Pa.
Ernest Sommer,	Portland, Ore.
Kellogg Speed,	Chicago, Ill.
John B. Walker, *Secretary*,	New York.

Because of the lack of facilities for the treatment of fractures in hospitals, the committee thought that its first duty was to formulate a minimum fracture equipment, then to bring this to the attention of all hospitals through the standardization committee of the college.

At the Chicago meeting of the college, the Board of Regents accepted the recommendation of the Fracture Committee, that to the minimum standard for hospitals eventually be added the following:

That all general hospitals be equipped to care for fractures.

That the minimum equipment for transport and emergency treatment be the following or its equivalent:

1. Thomas upper extremity splints.
2. Thomas lower extremity splints, with traction straps, slings and buckle straps.
3. Hodgen splints.
4. Coaptation splints, assorted sizes.
5. Cabot wire splints.
6. Straight pieces of wood of assorted length, width and thickness for splints.
7. Plaster-of-Paris bandages.

8. Some form of overhead frame for suspension.

9. Suitable *x*-ray apparatus, including a portable machine, if practicable.

It is carefully stated that the equivalent of the specified minimum will be accepted. This allows a suitable latitude to each institution in complying with the standard.

The committee also reported that it is highly desirable that one individual surgeon be responsible for the supervision of the care of fractures in each hospital service.

The committee also recommended that special record sheets be used for fracture cases.

The committee realized that because almost all fractures are first seen and treated by general practitioners, and that this will necessarily always be so, an educational movement to inform the general practitioner of fracture treatment was important. Consequently the committee has organized throughout the country local committees which have held meetings during the year for the discussion of fracture problems.

It is clearly evident that an increasing interest in fractures is aroused among surgeons through these committee meetings.

The New York and Philadelphia Committees are well organized. Some thirty or forty men are in attendance, monthly meetings are held, reports are made to the general committee, and these reports are mailed to other local committees for their information. The subject for discussion in the local committee is suggested partly by the general committee.

Thus an increasing number of facts bearing on general principles and the treatment of individual fractures is accumulating for future use.

It is hoped that this material, edited and arranged in simple form, may be eventually distributed by the College to all its members.

The committee, in addition to thus assisting the *graduates* in medical schools, has thought it desirable to seek to improve instruction given to *undergraduates* in medical schools throughout the United States and Canada. With this aim in view a question-

naire was sent to thirty-four medical schools. Returns have been received from the professor of surgery in thirty-two schools. All replies expressed approval at the investigation and promised coöperation with the committee. The following is a copy of the questionnaire:

1. Is instruction given in fractures?
2. In what school year given?
3. By whom; name of instructor?
4. How many exercises?
5. Is this instruction in systematic lectures?
6. Is it clinical?
7. Does any student himself care for fractures under supervision?
8. How much ground is covered?
9. Would you welcome suggestions from this committee regarding the conduct of fracture instruction?
10. Would you approve an attempt to standardize—simplify—such instruction in the medical schools throughout the country?

A subcommittee of the General Fracture Committee has been formed, called the Committee on Medical School Education. This committee consists of Drs. Darrach, Ashhurst and Scudder. This committee has made the following report, which will be sent to the professor of surgery in all Grade A medical schools, with the hope that it may prove helpful and constructive:

"After a careful study of the thirty-one replies to a questionnaire sent to various medical schools, it is realized that the amount of time spent on the teaching of fractures varies almost as much as does the content of these courses. Further, there is a wide variation in the facilities, both clinical and laboratory, in the different schools. The tendency in medical education apparently is against too great extension of courses and against the limitation of teachers to exact hours and content. It is the earnest belief of the committee, however, that there are certain fundamentals which every medical student should know. It therefore submits the following as a recommendation to be sent by the General Committee to all heads of surgical departments in the various medical schools:

Suggestions for the Teaching of Fractures and Dislocations. The General Fracture Committee of the American College of Surgeons suggests that in presenting the subject of fractures and dislocations to undergraduate medical students, the following principles be observed:

1. That each student should thoroughly understand the pathology of fractures, including the process of repair. That when possible the presentation of this subject should include animal experiments, the demonstration of specimens, both microscopic and macroscopic, illustrating the various stages.

2. That each student thoroughly understand the principles of the treatment of fractures as outlined in the syllabus adopted at a conference held in Boston in April, 1922, and published in the *Archives of Surgery* in January, 1923, vi, 172–194.

3. That each student have a thorough understanding of the use of splints for transport and emergency treatment, recommended by the General Fracture Committee, and have experience in their practical application.

4. That each student thoroughly understand the principles and practical application of traction and suspension.

5. That as much clinical experience and instruction be given as the clinical facilities and the curriculum of each school can afford.

The Fracture Committee believes that, with the suitable equipment of hospitals for the reception of fractures and their adequate treatment, coupled with an intelligent interest among practitioners of medicine in the subject of fractures (aroused by the activities of the local committee), and with a sensible educational program in force in the medical schools of the country, lasting good will be accomplished and a distinct advance made in the treatment of fractures.

The committee is now turning its attention to the end-results study of fractures, from which it is hoped much may develop.

I cannot refrain from saying simply one word at this time upon the very important relation of the Fracture Committee's activities to industrial surgery. The surgery of industry, the

surgery necessary to care for the worker and laborer in the United States and Canada, has attained huge proportions. Workmen's compensation laws in the United States date from 1911. Such laws are now in effect in forty-two states. Arkansas, Florida, Mississippi and North and South Carolina have not yet passed such laws. The law enacted by the legislature of Missouri was defeated by referendum. The enactment of compensation laws introduced a new factor into the industrial order in the United States. It made the employer directly responsible for injuries to workers while in his employ. A monograph, entitled "Workmen's Compensation Acts in the United States," has just been published by the National Conference Board under the editorship of Dr. Frank L. Rector, secretary of the Conference Board of Physicians in Industry.

The economic demand from the employer of labor, from the insurance companies, from the laborer himself, imposes upon the surgeon today a tremendous pressure to bring about, in the injured workman, as speedy and as complete a return to industry as is possible.

Traumatic surgery, of which the surgery of fractures forms a very important part, demands special training.

No one should assume the care of fractures who is not fitted to meet properly the very serious complications, sequelæ and attendant emergencies of fracture treatment.

The Fracture Committee hopes that its activities through the influence of the college may contribute to establishing sound educational opportunities, which will place the practice of traumatic surgery on the highest possible plane. A new era is opening to the surgeons of traumatism. The scope of such a specialty is far reaching and may well attract recent splendidly equipped graduates in surgery.

BONE TUMORS: MYXOMA; WITH REPORT OF THREE CASES

(SECOND PAPER)

By JOSEPH COLT BLOODGOOD, M.D.
BALTIMORE, M.D.

The first complete report was published in the *Annals of Surgery* in December, 1920 (vol. xxii, p. 713).

The point emphasized in this report was that every myxoma of bone on record recurred locally—and three of the cases reported died of metastasis. [A fourth subsequently died of metastasis to the lungs, and was reported in the *Annals of Surgery* for January, 1923.]

Myxoma Other than of Bone. Apparently myxoma growing outside of bone is less malignant both as to local recurrence and internal metastasis. I have 2 cases of pure myxoma of the breast in which the tumors were excised without exploratory incision, one by Dr. Tyler of Greenville, S. C., and the other by Drs. Walheimer and Saltzstein, of Columbia Hospital, Milwaukee, Wis. Both these patients are well, one more than three and the other more than five years since operation. These myxomas somewhat resembled in the fresh the myxoma of bone, but they were firmer, and when pressed did not exude a liquid tapioca-like material or break up into particles like vesicles of a thin mucilage consistency. Under the microscope these myxomas show the lymphoid and stellate cells, but the stroma takes a little of the eosin staining, and there is evidently more fibrous tissue than in the myxoma of bone.

The connective-tissue part of the so-called intracanalicular

myxoma of the breast differs from these two cases of pure myxoma in being less gelatinous, firmer, and under the microscope we rarely see the lymphoid or stellate cells. The characteristic feature of this adenoma of the breast is that the stroma takes the stain very lightly and resembles the stroma forming the envelope of the ducts and acini in the normal breast. These intracanalicular myxomas of the breast may recur when they are shelled out, but the recurrent tumors have always been benign and the patients have never died of metastasis.

The myxomatous part of the so-called mixed tumor of the parotid and neurofibroma is more like myxoma of bone, but in the gross rarely so gelatinous. But these two tumors do not show the same vitality in local recurrence when exploratory incision is made.

In the first paper on myxoma and in a recent paper on xanthoma[1] and in other contributions I have drawn attention to the danger of local recurrence when tumors, even benign tumors, and even encapsulated benign tumors, are enucleated or "shelled out." The best example is the mixed tumor in the region of the salivary glands. The percentage of local recurrence here is larger. This can be explained by the fact that surgeons fear injury of the facial nerve, and for this reason practice enucleation, or shelling out. Perhaps in the neurofibroma local recurrences take a second place, especially if the tumor is near a large nerve trunk. In the intracanalicular myxoma of the breast local recurrence is less frequently recorded, because most surgeons cut these tumors out, as taking away a little larger zone of breast tissue is not mutilating. However, in the myxoma of bone there is additional danger if one even cuts into the tumor—the danger of transplantation of tumor cells into the open wound.

Definite sarcoma not differing from any other type of sarcoma may develop in any of the connective-tissue tumors, apparently more frequently in a benign tumor containing myxomatous tissue. The most common sarcoma of the breast arises in the

[1] Archives of Surgery, May, 1924, vol. 8, p. 882.

intracanalicular myxoma. Why sarcoma is more frequently observed in neural fibroma than in the myxomatous tumor in the region of the salivary glands I cannot explain.

In one of the cases reported in my paper in 1920, in which the primary tumor was located in the astragalus, there was definite metastasis of pure myxoma to the tubercle of the tibia after amputation above the ankle-joint (Case II, Fig. 11).

Unfortunately, I have never been able to obtain the lung metastatic tumor in any of these cases of myxoma of bone.

Central Myxoma of Phalanges. In the older literature, which I have discussed in *Progressive Medicine* and in my paper on "Bone Cysts" in the *Annals of Surgery* for August, 1910, cases of central myxoma of the phalanges are reported. This group of tumors in the phalanges of fingers and toes have been constantly restudied since. Unfortunately, in not a single instance in which the original diagnosis was myxoma have we any sections to prove it. In all the recent cases in which we have tissue and sections none are myxoma—all are chondromas. Therefore, either the myxoma of the phalanges are less malignant or the older diagnoses were incorrect. On the theory of probabilities, myxoma should now and then be seen in the phalanges, because this tumor occurs where the chondroma does, and it is suggestive that it is a degeneration of a primary chondroma; but the practical fact remains that the few cases diagnosed myxoma, situated in the phalanges and curetted without chemical or thermal cauterization, have not recurred locally or given rise to metastasis. My colleague, Dr. Baer, of Johns Hopkins, curetted such a case some years ago and has recently sent me an *x*-ray, showing complete ossification. I remember distinctly the gross appearance in this case, and my written report strongly suggests a myxoma, but tissues and section have been lost.

For reasons which at present we do not know, all connective-tissue tumors arising from the soft parts and bone in the region of the phalanges of the fingers and toes are benign. We have only one example of sarcoma which was part of a multiple endothelial sarcoma of the phalanx of the great toe, tibia, and femur.

I am rather inclined to feel that a surgeon exploring a central tumor of a phalanx and finding the gross and microscopic picture of a myxoma would be justified in curetting with thermal (cautery) and chemical cauterization.

Central Myxoma of Metacarpals. What has been said of the phalanx applies to the metatarsal and metacarpal bones.

Tumors of the Carpus. Up to the present time I have never observed any type of malignant tumor of the carpal bones.

Central Tumors of the Tarsal Bones. My experience has been with the astragalus and os calcis only. The possibilities are bone cysts, giant-cell tumors and myxomas. The giant-cell tumor is the most common. There is no way of diagnosing it in the *x*-ray. If one explores a central tumor of the astragalus or os calcis and exposes a bone cyst, a giant-cell tumor, curetting with thermal and chemical cauterization is sufficient. All of the cases of which I have records are well. Dr. Prince, of Rochester, has had an unusual experience with this group. If, however, the central tumor suggested in the gross and in the frozen section a myxoma, I would immediately burn the wound with the cautery, clean up the leg above the ankle, and, with a new set of instruments, *amputate*, in order to avoid the local recurrence and perhaps death from metastasis recorded in Case II of my paper of 1920.

Central Tumors of the Long Pipe Bones. The problems of diagnosis here are easier, but the correct method of treatment for myxoma is not yet established, although one should be inclined to choose resection or amputation.

My studies up to date emphasize that the myxoma, either central or periosteal, is for practical purposes a sarcoma, with a much greater tendency to recurrence from wound transplantation and with a longer interval before death from metastasis. It is important to repeat here that all the older surgical pathologists as far back as 1840 wrote: "The myxoma of bone is a benign tumor, but it always recurs after operation." They did not observe their cases long enough to know of the metastasis. Many of my colleagues have not accepted this classification into central

and periosteal tumors of the long pipe bones, which, as a matter of fact, is not mine and not new. I am one of the few who look upon it as one of the fundamental facts established by the older observers of bone tumors.

More recent investigations allow some very positive deductions, based upon the age of the patient and on whether the tumor involved the diaphysis or epiphysis.

When the patient is under eighteen, and more certainly when under fifteen, a distinct, central tumor of the shaft of a long pipe bone is always benign. The majority of cases are benign bone cysts, a very few (less than 2 per cent) the benign giant-cell tumor, with now and then an isolated case of tuberculosis. I am looking now at the *x*-ray of a central tumor, due to tuberculosis, of the shaft of the tibia in a girl aged eleven years. The bone shell is intact.[1] In the twenty years I have record of but two other cases.[2]

If the central tumor is in the epiphysis and the patient is less than eighteen years of age, and more certainly if he is less than fifteen, the tumor is usually a giant-cell tumor, but may be a bone cyst or tuberculosis. This localization in the epiphysis is very rare for the bone cyst, and this age is very uncommon for the giant-cell tumor.

When the patient is an adult, and the central tumor involves the epiphysis, the chances are that it is a benign giant-cell tumor; but chondroma, myxoma, and sarcoma are possible. When the central tumor involves the shaft of an adult bone, one can almost exclude a bone cyst or giant-cell tumor, and one must think of myxoma, chondroma, myeloma, metastatic tumors, and the very rare sarcoma. I am able to report three additional cases of my own since my paper in 1920:

Case I is chiefly of interest because the recurrent tumor, in the shaft of the upper third of the femur, has resisted repeated cauterizations with plumber's soldering irons for a period of more than one year. Had the operator in this case been familiar with

[1] See Fig. 60, Progressive Medicine, December, 1900, p. 2080.

[2] See Journal of Radiology for March, 1920 (Figs. 37 and 38).

the use of the cautery, and the fact that a central tumor in the shaft of a long pipe bone of an adult is rarely a giant-cell tumor or a cyst, this thermal cauterization might have been employed at the time of the first exploration and curetting, in June, 1922, instead of after the second curetting for recurrence, in July, 1923.

Case II suggests that it is possible to recognize the central malignant tumor from the clinical history, in the *x*-ray and by palpation, and perform amputation or resection without an exploratory incision.

In Case III, the operator, Dr. Rockey, was not suspicious of myxoma or a central lesion other than cyst or giant-cell tumor in the lower end of the femur, but at the exploratory incision he recognized in the gross an unusual tissue, and for this reason used the hot soldering irons. This patient is clinically well, with a good functioning limb, nine months after operation.

Case I.—Pathol. No. 23423. *First observed in July*, 1923. *Central myxoma of the upper shaft of the femur, extending to the greater trochanter; recurrent one year after curetting without chemical or thermal cauterization.*

In this case I have the tissues of the first operation, from the operations for recurrence, and of the last recurrence (September 20, 1924). The tumor is still a pure myxoma, both in the gross and microscopic. (See Figs. 1, 2, 3 and 4.)

The next most important fact in this case is that in June, 1922, two years and four months ago, on the diagnosis of a bone cyst, the bone shell was opened and the contents curetted out. There was no chemical or thermal cauterization.

On July 23, 1923, because of recurrence, shown by palpation, *x*-ray, and pain, a second operation was performed by the same surgeon, and because of the profuse hemorrhage after curetting the wound was packed. A few days later this wound was thoroughly cauterized with hot soldering irons.

August 8, 1923, less than two weeks after the second operation, the patient was admitted to St. Agnes' Hospital and the open

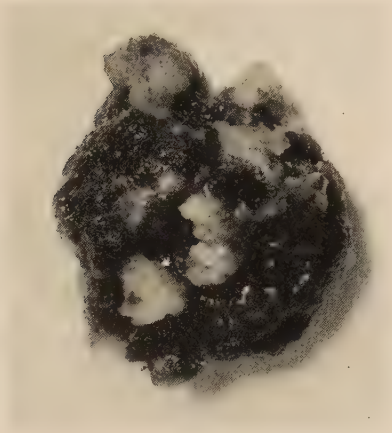


FIG. 1.—Case I. Pathol. No. 33423. Gross tissue floating in a hemorrhagic fluid. The white tapioca particles mixed with the tumor tissue stained dark by blood resemble the tissue in Fig. 11.

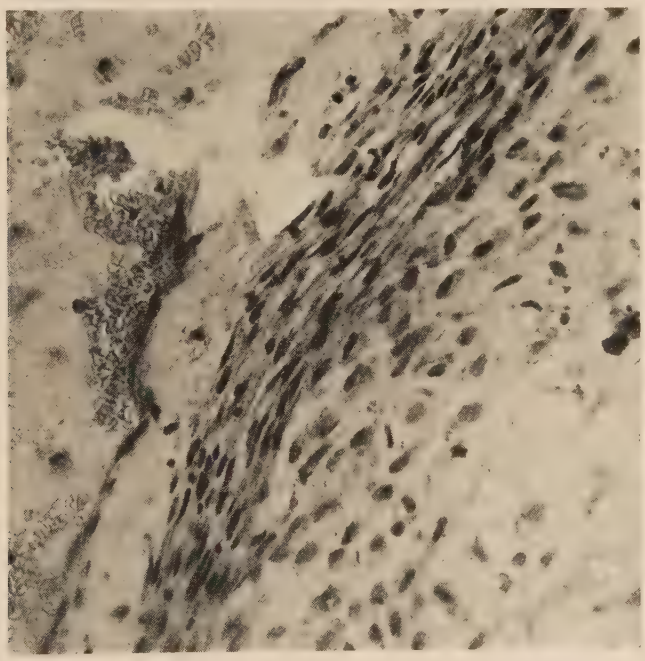


FIG. 2.—Case I. Pathol. No. 33423. From the first operation curetting in June, 1922. The hemorrhagic part is separated from the myxomatous tumor by condensed cellular tissue, suggesting spindle-cell sarcoma.

wound again cauterized with the thermal cautery. This cauterization has continued at intervals for now more than one year. In spite of this, at the last operation (September 20, 1924) pure myxomatous tissue was found in a bone cavity below the great trochanter.

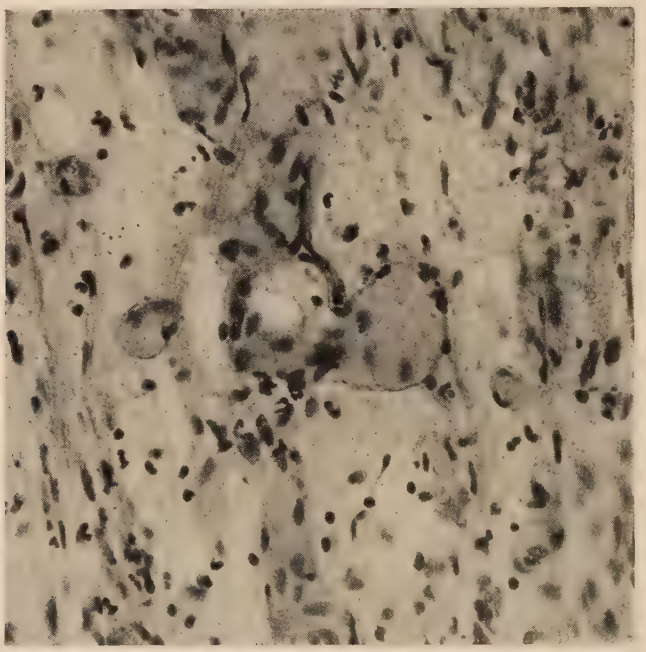


FIG. 3.—Case I. Pathol. No. 33423. Recurrent tumor tissue in a bone cavity. Observe the bloodvessels and blood mixed with myomatous tissue.

The patient still walks with a cane, and for at least one year has been able to continue his work as a postal clerk. The cauterizations every three to six weeks, under gas anesthesia, have kept him in the hospital from three to five days. When I exhibited this patient before the American Surgical Association, in May, 1924, the open wound was clean and there was no gross or microscopic evidence of recurrence. We have, therefore, demonstrated the failure of the thermal cautery and the deep *x*-ray to check or

destroy the local growth of a recurrent myxoma. At the present time there are no signs of metastasis to the lungs.

Clinical history taken before the operation in June, 1922. First, there is a distinct history of a direct trauma to the region of the great trochanter of the right femur, in a white male, aged forty-nine years, whose occupation is that of a mail clerk. The

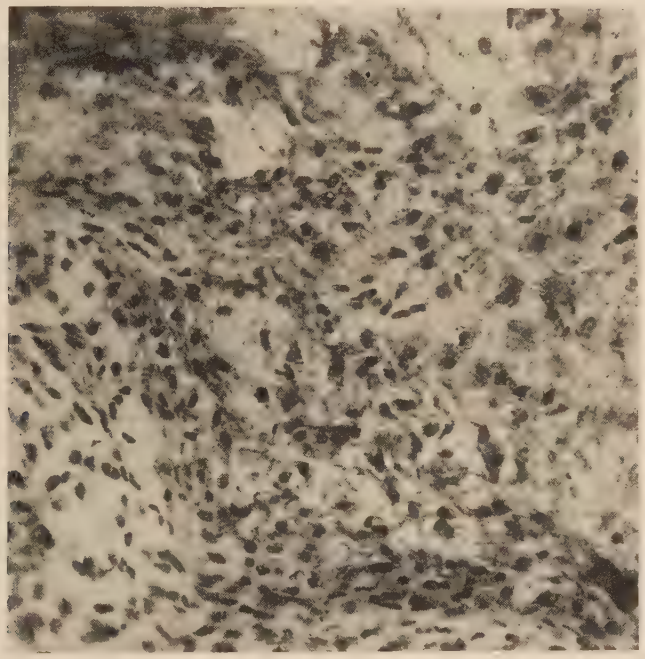


FIG. 4.—Case I. Pathol. No. 33423. A section from a recurrence two months later than Fig. 3. Typical myxomatous cells, but less intercellular myxomatous tissue.

pain after this trauma lasted but a few days, and there was an interval of almost two years before the occurrence of severe pain in the region of the great trochanter, upper end of the femur and sacro-iliac joint, and this pain had been present for more than one year before the operation in June, 1922.

Pain in Myxoma. We have not a sufficient number of cases to come to any definite conclusions, but so far my records show

that an intense, persistent pain, in a bone lesion in which the *x*-ray shows an intact bone shell, is very suggestive of myxoma or rare sarcoma. I have, however, observed it on a few occasions in the giant-cell tumor, very rarely in the bone cyst.

X-rays and Palpation. The surgeon who operated in June, 1922, reports that he could palpate a swelling below the great trochanter on the outer lateral surface of the right thigh with

FIG. 5.—Case I. Pathol. No. 33423. Appearance of the wound August 25, 1923. There had been three intensive burnings with the soldering irons, in attempting to destroy all the soft parts about the recurrent tumor and all the cavities containing tumor in the trochanter and shaft.

a distinct bone shell. The *x*-ray report notes a definite central lesion occupying the greater trochanter, extending to the shaft about the size of a hen's egg. The outer bone shell is thin, but the shaft of the femur and the great trochanter are only partially invaded. Unfortunately, this *x*-ray film is lost.

Clinical and X-ray Diagnosis. The one made at that time by the surgeon and the roentgenologist was *bone cyst.*

From my experience the location and the age almost ruled out

the bone cyst and the giant-cell tumor—too old for the bone cyst and an unusual position for the giant-cell tumor. One should have thought of myxoma, chondroma, or a possible sarcoma. The history of the intense pain also suggested the more malignant types—myxoma and sarcoma. For this reason the patient should have been prepared for the immediate cauterization with a heavy soldering iron.

FIG. 6.—Case I. Pathol. No. 33423. *X*-ray October 10, 1923. The patient is apparently well; the last section showed no tumor. The new bone formation outside the shaft and trochanter is largely the result of two previous operations and repeated cauterizations.

Gross Pathology of First Operation. The operator informed me that there was no distinct connective-tissue lining as is usually found in the bone cyst, and no red, granular, vascular tumor so characteristic of the giant-cell tumor; but a viscid, whitish, thick fluid containing masses like tapioca. This almost describes the myxoma and resembles the contents of the cyst found at the second operation.

Recurrence after the First Operation. The wound healed and the patient was relieved of pain for a few months. Then there were similar recurrent pains which radiated from the great trochanter toward the sacrum around the hip and down the thigh. A few months later a mass could be palpated and recurrence was diagnosed by the original operator.

FIG. 7.—Case I. Pathol. No. 33423. *X*-ray September 25, 1924, before the last operation for recurrence. Compare with Fig. 6. There is an area of bone destruction and in this cavity tumor tissue was found.

X-ray of Recurrence (July 1, 1923). I examined this patient July 10, thirteen months after the first operation, at least eight months after the recurrence of the pain and three or four months after the appearance of a visible and palpable swelling in the scar, and I examined this *x*-ray, and further *x*-rays were taken by Dr. Kahn in my office. Unfortunately, they have been mislaid or lost. (I took them with me to another city for a demonstration and returned without them.) But this *x*-ray and the ones which we have taken subsequently, and which are reproduced

here, differ very little from the description of the *x*-ray before the first operation, except that the cyst is larger; no marked destruction of the shaft appears in the *x*-ray. There is nothing in any of these *x*-rays to distinguish the type of tumor—it could be a cyst, a giant-cell tumor, or a chondroma. In the *x*-ray taken after the first operation I was surprised to find a reformation of the bone shell, which had been removed, with considerable evidence of new bone formation toward the great trochanter and a cavity the size of a hen's egg, as described in the first *x*-ray. The larger part of the cyst was outside the trochanter and shaft, and expansion carrying with it the envelope of bone shell. The swelling at the time of my examination was fully the size of a grape-fruit (12 cm.), and when I dictated this note (July 10) the patient was free from pain, there was no tenderness, and very little limitation in flexion and rotation at the hip-joint. The patient was walking without crutch or cane and able to work. There were no signs of recurrence in the soft parts, and, to repeat, a distinct reformation of the bone shell. For this reason I was rather inclined to the diagnosis of chondroma which had liquefied, because in myxoma I rather felt from my previous experience that there would be recurrence in the soft parts. This case shows how difficult it is, when the number of cases is small, to be certain of any conclusion based upon such previous experience.

The patient returned home, and because of intense pain the original operator explorated, evacuated the contents of the bone shell, and, as stated before, packed for hemorrhage. A few days later, at my suggestion, he thoroughly cauterized the wound with soldering irons.

Pathology of Second Operation. The material sent to the laboratory was a thick, viscid fluid stained with blood and filled with small and large masses, which consisted of opaque, gelatinous tissue not unlike tapioca, imbedded in tissues stained with blood. Fig. 1 is one of the larger pieces. Figs. 2, 3, and 4 show the microscopic appearance: certainly a myxoma, and some pathologists would say a myxosarcoma. Fig. 5, taken August 25, 1923, after three intensive cauterizations at St. Agnes' Hospital,

shows the open wound. Fig. 6 is an *x*-ray taken October 20, 1923, after repeated cauterizations, at a time when the patient showed no evidence of disease in the wound. Fig. 7 is an *x*-ray taken September 25, 1924. You will observe that the area of destruction has appeared in the shaft below the trochanter, and in this cavity we found gross and microscopic evidence of tumor tissue. To repeat, there is no evidence of metastasis to the chest. The patient is free of pain, walks with a cane, and continues at work in the post-office department.

Discussion of Case I. I have called attention to this in previous communications, and the records of the bone tumors accumulated in the past few years emphasize the importance of this statement. No surgeon should explore a bone tumor without being prepared to cauterize the wound. The thermal cautery is far stronger than any chemical cauterization, and, fortunately, the best cautery is the cheapest—the plumber's soldering irons.

This case cannot be looked upon as a fair test of the use of the cautery in the treatment of myxoma. We shall have to follow the result in the third case. It does, however, confirm the conclusions of the previous papers on myxoma—its tendency to local recurrence. I am convinced that no local disease has ever received a more thorough treatment with the thermal cautery and the deep *x*-rays than in this case.

At this time (September, 1924) resection was out of the question, and amputation, of course, means hip-joint disarticulation. From previous experience an amputation does not promise protection against metastasis. We have given this man a useful limb for two years since his first curetting.

CASE II.—Pathol. No. 34522, *observed January 27, 1924. Central myxoma of the lower end of the femur, involving the shaft and the epiphysis. Pure myxoma type. First, deep x-rays, then amputation. Result, September, 1924, eight months, well.*

This is the first time that I have deliberately made the diagnosis of a possible myxoma and amputated without exploratory incision.

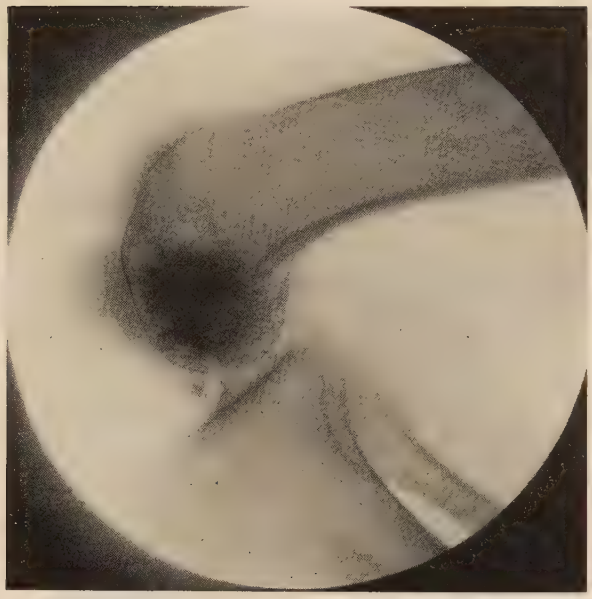


FIG. 8.—Case II. Pathol. No. 34522. *X*-ray taken in 1920 and diagnosed a benign ossified lesion.

FIG. 8a.—Case II. Pathol. No. 34522. *X*-ray taken in 1920 and diagnosed a benign ossified lesion.

The next most interesting point is that this is the first case in which I have an *x*-ray of the bone taken before the development of the tumor, and this bone showed evidence of a preëxisting and apparently healed central lesion of the epiphysis.

Previous History. In November, 1920, three years and ten months ago, this patient, a white female, then aged thirty-seven

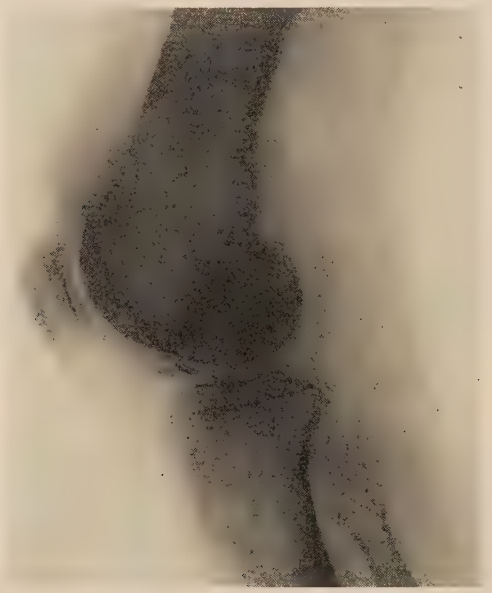


Fig. 9.—Case II. Pathol. No. 34522. *X*-ray, lateral view of myxoma of lower end of femur, shaft and epiphysis, with remains of the old lesion in the condyle shown in Fig. 8. Note there is no expansion; minute perforation of cortical shell; evident tumor shadow with, perhaps, some bone formation outside the cortical shell, both anteriorly and posteriorly.

years, came under my observation and was operated upon for a benign tumor of the breast. At that time, because of pain in the right leg in the region of the knee-joint, an *x*-ray was taken (Fig. 8). This was interpreted as a healed and ossified area of tuberculosis or a rare ossified bone cyst. I explored the knee-joint, found a little fluid, but a normal synovial membrane and cartilage. Microscopic sections showed no disease. This patient

had been complaining of this joint for ten months and has been using a cane for four months.

In the *Journal of Radiology*, March, 1920, I recorded a tumor of the lower end of the femur—as far as I know the first on record—in which an *x*-ray made July 1, after an injury, was negative. As pain and disability continued, further *x*-ray pictures were

FIG. 10.—Case II. Pathol. No. 34522. Anteroposterior view, not as clear as Fig. 9. The remains of the tumor shown in Fig. 8 are seen here in the internal condyle. This *x*-ray immediately should make one suspicious of myxoma or sarcoma. The disease has broken through the bone shell without expansion.

taken. Within five weeks an area of bone destruction was found in the external condyle and the area increased in size up to September 1, when the lesion was explored and amputated on the diagnosis of a central sarcoma of the malignant hemorrhagic-cystic type. This patient is well more than five years and the diagnosis has been changed, by a majority vote of Codman's registry, to a benign giant-cell tumor. The interesting

part of the case, however, was a series of *x*-rays after the trauma with negative findings. The interesting and unique part of the case reported here is the *x*-ray more than three years before, picturing an apparently healed lesion.

FIG. 11.—Case II. Pathol. No. 34522. Longitudinal section through lower end of femur, formalin hardened specimen. Compare with Fig. 10 (*x*-ray).

Present History. Whenever this patient wrote me she complain of pain in this knee. We were all inclined to look upon it as neurasthenic. For the past three months the pain has been almost continuous, of greater intensity; there has been some

contraction of flexion and the patient has had difficulty in walking.

Examination. There is the expression of pain and fear. The involved knee is slightly flexed; there is visible swelling of the lower end of the thigh, which, on palpation, is not fluid in the joint but swelling of the soft parts. The only benign lesion of

FIG. 12.—Case II. Pathol. No. 34522. Photograph of specimen to show soft-part tumor in the popliteal space from perforation of the bone shell. The piece to the right is some muscle and fascia, sliced from the bone, exposing tumor tissue at *x*.

the bone presenting so much pain and tenderness was a central lesion of the upper end of the humerus in a boy, aged sixteen years, reported in the *Journal of Radiology* for March, 1920. This patient is well after curetting only, and there is a difference of opinion in Codman's registry as to whether it should be considered a sarcoma or a variant of the giant-cell tumor.

The point which I wish to emphasize again here is that extreme pain and tenderness in a bone lesion without fracture and without evidence of inflammation is very suggestive of malignancy.

X-rays (Figs. 9 and 10). The lateral view is better. Both views show a central light area, involving the shaft and the epiphysis, invading the old healed area, recorded in the *x*-ray (Fig. 8) more than three years ago. The age (beyond twenty) is against bone cyst; the involvement of the shaft as well as of the epiphysis is against giant-cell tumor. Then we evidently see cortical destruction, with a soft-part tumor shadow outside the cortical layer. There is also practically no expansion. In my experience this *x*-ray should be looked upon as a malignant tumor, and this case shows that the pure myxoma may give the same *x*-ray picture as the central myxosarcoma or chondrosarcoma reported and illustrated in the *Journal of Radiology* for March, 1920.

Treatment. We kept this patient at absolute rest in bed, except when she came to the office, for *x*-ray treatment, on crutches. Intensive radiation was given by Dr. Max Kahn from January 7 to 21. Intravenous salvarsan was given by Dr. Collenberg, although the Wassermann was negative. The *x*-rays of the chest and all other laboratory examinations were negative. As there was no improvement of the symptoms amputation was performed July 23, 1924.

Pathology. Fig 11 pictures the myxomatous tissue in the preserved formalin specimens in the fresh gelatinous material exuded from the cut surface. Observe in the internal condyle the old ossified lesion infiltrated by the tumor. The shape line of demarcation between the tumor and the marrow and the minute perforations of the cortical shell. Fig. 12, at *x*, shows a soft-part tumor outside the bone shell in the popliteal space, which we can see in the *x*-ray in the lateral view.

Microscopically. This is a pure myxoma. As a matter of fact, there is no difference between a pure myxoma and a myxosarcoma: Both seem to have the same tendency to local recurrence; perhaps death from internal metastasis is later in the pure myxoma.

Result in Case II (October 21, 1924). Nine months after operation the patient is well; no metastasis to the chest.

CASE III.—Pathol. No. 34618. *Central myxoma of the lower end of the femur. Operation: Curetting with hot soldering irons by Dr. E. W. Rockey, of Portland, Ore. Result: October, 1924, nine months, apparently well.*

FIG. 13.—Case III. Pathol. No. 34618. *X*-ray December 29, 1923, before operation. Note involvement of shaft and epiphysis; little or no expansion; suggestion of perforation anteriorly. Compare with Fig. 14, after operation.

I am indebted for the record *x*-rays, and tissue in this to Dr. Rockey, and, as far as I know, this is the first central myxoma to be attacked primarily with the thermal cautery. The tissues sent to my laboratory from this operation show pure myxoma.

X-rays before and after Operation. In this case I have an *x*-ray dated December 29, 1923, sent me by Dr. Rockey, and an *x*-ray

taken in my office by Dr. Kahn, dated August 14, 1924. The interesting question is, can we tell the shadow of recurrence from the shadow of fibrous healing without ossification?

Let us compare the lateral view before operation (Fig. 13) and eight months after operation (Fig. 14). The chief difference is that the cavity, or light area, in the lower end of the femur

FIG. 14.—Case III. Pathol. No. 34618. *X*-ray August 14, 1924, seven months after operation. Compare with Fig. 13. The central light area has now a more distinct ossified circumscribing shell. The perforation shown in Fig. 13 has ossified. All bones show slight osteoporosis. Compare the patellæ in Figs. 13 and 14.

and epiphysis is outlined in the picture after operation much more distinctly. In fact, the line of demarcation is as distinct as the cortical layer itself. The picture taken before operation shows a break in the cortical layer at the junction of the epiphysis and the shaft just beneath the patella.

Nevertheless, the cavity which contained the tumor, and which was burned out with the soldering irons, is still unossified.

The comparison of the anteroposterior views (Fig. 15) before operation and (Fig. 16) after operation, as compared with the normal right leg after operation (Fig. 17), shows that the portion

Fig. 15.—Case III. Pathol. No. 34618. Anteroposterior view. The shadow of the patella overlaps the central light area in the external condyle. The line of demarcation between the tumor shadow and the marrow and shaft shadow above is different from that of any bone cyst or giant-cell tumor. Compare with Fig. 16, after operation.

of the cyst extending up into the shaft from the external condyle suggests ossification. In the pictures before operation (Figs. 13, 14 and 15) the internal condyle is apparently not involved, while after operation there are light shadows, suggesting either that

the cancellous bone of the internal condyle was removed at the operation or is the seat of osteoporosis from non-use. In Figs. 14 and 16 the left knee-joint, after operation, shows distinct osteoporosis from non-use as compared with Figs. 13 and 15. of the affected limb before operation and Fig. 17 of the unaffected limb after operation.

FIG. 16.—Case III. Pathol. No. 34618. *X*-ray August 14, 1924, after operation. Most marked feature is osteoporosis; some ossification in shaft. No evidence of perforation.

I have studied this question before in a few other cases, and I find that in the majority it is impossible, at first, to distinguish in the *x*-ray light shadows of definite recurrence from light shadows due to fibrous tissue without bone or osteoporosis from non-use. I had an opportunity, some three years ago, to compare 2 cases of giant-cell tumor in the upper epiphysis of the humerus; both submitted to curetting, 1 without chemical or thermal cauterization, the other with. At the end of one year the *x*-ray shadows are apparently identical. The opening

in the bone shell through which curetting had been done appears ossified in the *x*-ray in both. Yet, after this, recurrence took place in 1; the first indication of which was definite local swelling, and *x*-ray showing destruction of the bone shell. I have just seen an *x*-ray of the other case, more than two years after curetting, and the cavity is not yet completely ossified.

FIG. 17.—Case III. Pathol. No. 34618. *X*-ray August 14, 1924, of the unaffected right leg, for comparison.

So in this patient of Dr. Rockey this apparent ossification of the bone shell cannot be looked upon as positively excluding recurrence, nor the persistence of the light area as an indication of recurrence. This patient will have to be carefully watched, and if one sees evident destruction of the bone shell or enlargement of the light shadow this must be looked upon as a sign of recurrence.

Clinical Note. This patient was examined in my office August 14, 1924, by Drs. Stewart and Cohn, and *x*-rays were taken by

Dr. Kahn. It is noted that the patient is a white male, aged forty-nine years. Two years before the operation, in January, 1924, without a definite history of rheumatism and while driving his automobile, his left leg felt cramped and he was conscious of pain in the region of the left knee. As this continued he consulted physicians and was treated for rheumatism. When he came under Dr. Rockey's care, in December, 1923, an *x*-ray was taken. Dr. Rockey wrote to me that from the *x*-ray he was

FIG. 18.—Photograph of plumber's soldering irons and gas furnace. Total cost $35.

inclined to the diagnosis of a bone cyst or a giant-cell tumor. The patient complained to him of pain for a period of two years and a recent limp.

Operative Pathology. (Dr. Rockey.) On exploring the bone over the tumor area the shell was so thin that it was broken by a retractor. A window was made in the bone shell and the cavity curetted and *thoroughly burned out with hot soldering irons* of various sizes. The wound healed *per primam.*

Microscopic Pathology. Sections sent to me from this tissue showed myxoma.

FIG. 19.—The different types of soldering irons.

Discussion. What shall we do if this tumor recurs? Resection is out of the question. Even with a successful bone transplantation to replace the lower end of the femur the resultant limb is never as useful. It is therefore a question between further curettings with the soldering irons and amputation. Even before my experience in Case I, I urged amputation in this case of

Dr. Rockey's. I practised it in Case II without exploration. Now that Dr. Rockey's patient is doing so well, I have no evidence that he runs greater risk of metastasis by further delay if there is a recurrence. However, when there are clinical and *x*-ray signs of recurrence the evidence suggests amputation as the operation of choice.

Conclusions in Regard to Central Myxoma. (September, 1924.) If we leave out the possible central myxoma of the phalanges and metacarpals and consider only the long pipe bones and my one example in the astragalus, the conclusions made in December, 1920, hold good today. The only cases of myxoma or myxosarcoma which have been permanently cured have been subjected to amputation or resection without exploratory incision.

Every myxoma explored has recurred locally, except this Case III of Dr. Rockey, in which the soldering irons were used and in which it is not yet one year since operation.

At the present writing I would choose resection without exploration, if possible, with bone transplantation. This might have been done in Case I, reported here, at the first operation. When resection and bone transplantation would not leave a limb with good function, primary amputation, as in Case II, impresses me as the operation of choice.

There will be cases in which resection and amputation can be performed without exploration, because of the localization of the lesion and the character of the *x*-ray.

Undoubtedly, in many cases, one must explore. Then one should always have ready the cautery, the pure carbolic and alcohol, the 50 per cent solution of zinc chloride and a technician to make a frozen section and someone who can interpet it under the microscope. From my recent experience in cases in which I have actually seen the gross tissue, or in which I have obtained a clear description by the operator, I am impressed that all of them, or the majority of myxomas of the bone, have a definite gross appearance so characteristic that any surgeon who really

studies his gross pathology would be able to recognize it at the exploratory incision.

Is Curetting with Thermal Cautery Justified in the Myxoma? Until Dr. Rockey's patient is followed for some years and shows no evidence of recurrence, one should not employ this method, but at once resect or amputate.

Chondroma. I have called attention to the tendency of all chondromas to recur if the tumor is explored or removed piecemeal, and a piece of tumor cartilage left in the wound. This tendency is apparently almost as great as in myxoma, but up to the present time, although some of the patients have ultimately lost their limbs and a few have died of the local growth in the region of the vertebræ, none have succumbed to metastasis.

Brief Note on the Literature of Myxoma. I reviewed the scanty literature in my article in the *Annals of Surgery* for December, 1920, but at that time I had not read my colleague, Dr. Cotton's paper, which I would like to briefly abstract here.

In the *American Journal of Roentgenology*, 1918, vol. v., Nos. 2 and 4, Cotton and McCleary, of Baltimore, report an example of a myxoma involving the shaft of the upper end of the femur, in a female aged thirty-five years. There were two exploratory operations between November, 1915, and the amputation of the hip-joint, November, 1916. In the first paper there are excellent *x*-rays and photographs of the gross.

In their second report, in the same journal, vol. vi, No. 12, p. 594, the authors report that this patient lived in comfort for two years, then there was evidence of recurrence in the groin and metastasis to the lungs, and death took place in April, 1919, almost four years after the first exploratory incision and two and a half years after the amputation. A marked feature of this case was the extensive involvement of the shaft of the femur, and at the time of amputation both a central and periosteal involvement.

In the second paper a second case is recorded, involving the

shaft of the femur below the trochanter, in a male, aged forty-five years. The duration of the pain before operation was two years. The real lesion was not revealed until the *x*-ray was taken. An exploratory incision was made and the characteristic features of a myxoma exposed. The tumor was removed with chisel and curette. The wound healed in four months. There were signs of recurrence in six months, then amputation of the hip was performed. The patient succumbed in one year.

DISCUSSION

Dr. W. B. Coley, New York:

I feel that there is very little to be added to what Dr. Bloodgood has already said. In the discussion of Dr. Lewis' paper, I think it might be of interest to the members of the Association to very briefly review my own cases of sarcoma of the spine. Since 1895 I have observed 17 cases of sarcoma of the spine and sacrum, in all of which the disease had reached the inoperable stage at the time of first observation; in addition, there are 2 cases treated by other surgeons under my direction. As regards the sex, 10 were females and 9 males. A study of the locality of the tumor in my personal series shows 2 cervical, 7 dorsal, 7 lumbar and 3 sacral. A microscopical examination was made in 17 of the 19 cases, showing 8 giant-cell, 4 spindle-cell, 2 mixed-cell, 2 round-cell; and in 3 cases an *x*-ray diagnosis was made. The ages ranged from ten to sixty years; 8 (or 47 per cent) of the 17 cases remained alive and well from three to twenty-two years; 1 died of a recurrence at the end of six years; all of these were treated with toxins alone, with the exception of 2 cases, in which radium or *x*-ray was used in conjunction with the toxins. The microscopical diagnosis was: Mixed-cell in 1 case, spindle-cell in 1, round-cell in 1, giant-cell in 2, and in 3 cases an *x*-ray and clinical diagnosis only was made. A brief history of some of these cases may be of interest:

Case I.—D. G., male, aged twenty years, was always in good health until the latter part of 1901, when he developed a swelling in the middorsal region; this grew rapidly; partial paralysis of the lower extremities developed a short time later and he was sent to the Montefiore Home for Incurables. I saw him at this institution in consultation with Dr. V. P. Gibney, in February, 1902. At this time he had a very large tumor occupying at least five or six of the dorsal vertebræ; he had complete paralysis of the bladder, rectum and lower extremities, and had lost fifty pounds in weight. The condition seemed to be absolutely hopeless. He was put upon the mixed toxins of erysipelas and Bacillus prodigiosus, without any other treatment; this was kept up for four months. His improvement was immediate and remarkable; in a few months he was able to walk about with a plaster cast. His recovery was complete; a few years later he married, and now has two children. A recent examination (March, 1924) showed the patient to be in excellent condition. In this case a microscopical examination was made by Dr. Harlow Brooks, of the Bellevue Hos-

pital laboratory, who pronounced it a round-cell sarcoma with many atypical giant cells.

Case II.—Baby W., male, aged two years and nine months, was brought to me in March, 1911, by his father, who is a physician in Virginia. The father, while holding the child in his arms, was thrown out of a wagon, in February, 1911. While nothing was noticed at the time, the spine of the child was undoubtedly injured. One month later he began to get weak, and in the latter part of March some swelling was noticed, about two inches above the upper border of the clavicle, on the right side. This increased rapidly in size and the patient continued to lose power of the muscles. The condition was regarded as infantile paralysis. There was marked muscular tremor of both hands and almost entire loss of power in the arms; the patient could not move himself at all, neither could he hold up his head. An exploratory operation was done by Dr. Stuart McGuire, of Richmond, Va., which revealed the presence of a well-defined tumor springing from the laminæ and transverse processes of the vertebræ. A piece of tissue was examined microscopically and pronounced fibrosarcoma. The toxin treatment was begun in July, 1911, and kept up for one year. The child's weight steadily increased during the treatment; he regained considerable power; was able to move his hands and legs, and could hold up his head. Dr Ewing, who made a microscopical examination in March, 1922, reported as follows:

"I am unable to offer a positive and exact diagnosis. It is certain that you deal with a malignant tumor which may very well be called sarcoma. I am inclined to think it is either an endothelioma secondary to the cerebral growth or possibly a neurocytoma derived from misplaced nerve tissue in the cranium."

In May, 1921, the boy's father decided to take him to Dr. Harvey Cushing for a laminectomy, who operated and found what he considered "a very extraordinary tumor (which Wolbach thinks is a ganglioneuroma) plastered all along the spine, though entirely extradural." A later report from Dr. Wolbach classified the tumor as a neuroblastoma of an undifferentiated type.

Case III.—H. B. H., male, aged thirty-eight years. In February, 1895, the patient began to lose flesh and strength; soon after he had pain in the lower part of the spine over the sacrum, the pain extending down the legs, more marked on the right side. On May 2 he was sent to St. Luke's Hospital, where he was examined by Dr. F. P. Kinnicutt and other members of the staff; the opinion of all was that

he had an inoperable sarcoma of the sacrum and pelvis. I saw the patient in consultation on May 10, 1895, and advised a brief trial of the toxins. The injections were made directly into the buttocks. At the end of one week the pain had almost entirely subsided and the lameness was much improved. At the end of seven weeks the patient had gained twenty eight pounds in weight and was in excellent condition. He remained well for seventeen years, when, in an accident, he received an injury to his skull which resulted in his death.

Case IV.—D. C., male, aged thirty-five years, operated upon in the latter part of July, 1910, by Dr. William J. Mayo, who removed the coccyx and the lowest sacral vertebra with the tumor, which grew from the periosteum of the coccyx and fifth sacral vertebra; the sacrococcygeal juncture was penetrated by the growth and the operation was an extensive one. The patient was put upon the mixed toxins as a prophylactic. In March, 1911, when he again reported to me, his weight was normal and the pain, which had been continuous since some time before the operation, had greatly diminished. He remained in good health until the early part of October, 1912, when he found himself suddenly unable to empty either bladder or rectum. He was again put upon the toxins and again showed marked improvement. The patient remained well for about five years, when the disease again recurred, proving fatal in 1917.

Case V.—Adult female, seen in consultation at the Mt. Sinai Hospital in August, 1918, at which time she was suffering from a tumor of the dorsal spine which had caused paralysis. Clinical and *x*-ray diagnosis, inoperable sarcoma. The patient received the toxins at the Mt. Sinai Hospital, under my direction. She made a complete recovery, and when last heard from, two and a half years later, she was up and about and doing her own housework.

Case VI.—H. L. V., adult female, was referred to me in June, 1913, with a history of an incomplete operation having been performed at the Mayo Clinic for a giant-cell sarcoma of the sacrum and coccyx; incomplete removal of the two lower segments of the sacrum and the coccyx; twenty-four injections of the mixed toxins were given; the highest reaction was 105°. The tumor mass gradually softened and disappeared, and the patient made a complete recovery. A recent letter from her husband (in March, 1924) states that she is still well.

Case VII.—Mrs. S., aged fifty-six years, had a tumor in the region of the middorsal vertebræ for about nine months before operation.

About a year later a recurrence took place and she was put upon the mixed toxins in February, 1895. Immediate improvement was noticed and the patient was well when last heard from, eight years later.

One of the cases of this series has a special bearing on the question of whether or not a giant-cell sarcoma may produce metastases:

Case VIII.—T. D., female, aged twenty years, was always in good health until September 3, 1920, when, while getting off a moving train, she was thrown to the platform, striking on her left side, in the region of the level of the scapula. There was no pain nor numbness, but shortly after the patient had what she described as "feelings or twitchings;" the latter persisted, and in about three months had become so much worse that she was not able to sit in one position for any length of time. She was treated for rheumatism for three months, during which time she continued to grow worse and was finally ordered to bed for a week; no improvement was noticed at the end of this time; she began to have feelings of numbness in both feet, which gradually extended up the legs, until finally she was unable to walk. In September, 1921, she consulted Dr. Sidney A. Twinch, of Newark, who referred her to Dr. William Sharp, of New York. The latter operated on her September 12, 1921, finding a large tumor in the mid-dorsal region of the spine, which he partially removed, but owing to the severe shock and loss of blood it was impossible to do a complete operation. Microscopically, the tumor was pronounced a giant-cell sarcoma. At this time there was complete paralysis of the bladder and rectum, as well as of the extremities; there was no sensation below a line about two inches above the navel; the tendo-Achilles of both legs was markedly contracted, so much so that her toes pointed almost straight downward below the line of the tibia. Examination of the upper portion of the spine showed marked protuberance in the mid-dorsal region, the central portion of which corresponded to about the middle of the shoulder-blade. Physical examination by myself, on December 21, 1922, showed the patient to be paralyzed from the navel down; she had several large bedsores on her back; her general condition as regards nutrition and appearance seemed normal; no loss of weight. She was admitted to the Memorial Hospital on March 9, 1923, and discharged on March 24. While there an *x*-ray examination was made by Dr. Herendeen, who reported "stereoplates of the lumbar spine reveal the presence of a hard bony mass in the 10th and 11th vertebræ and ribs on the left side; the adjacent articulations are involved by

pressure, no evidence of active bone destruction is seen, but there is evidence of sclerotic changes; it is difficult to determine accurately the nature of the original process." The radium pack, 7202 mc. hours, was applied at 10 cm. distance over the dorsal spine. In addition she received a few doses of the toxins at the hospital, and was told to have the injections continued after her return home. This, however, she refused to do. She gradually grew worse, and in the latter part of 1923 and the early part of 1924 she developed metastases in the mouth and throat, ending in death in March, 1924.

The 2 cases that were treated by other surgeons are:

Case IX.—Case of Drs. Miketta and Oliver, of Cincinnati: Female, aged sixteen years. *X*-ray and clinical diagnosis: Sarcoma of the cervical spine (no microscopical examination was made). The patient made a complete recovery under the toxins, and is well fourteen years later.

Case X.—Case of Dr. Harmer, of Boston: A young male, with a recurrent inoperable sarcoma of the lumbar spine, with partial paralysis; complete recovery under the toxins alone; patient well eleven years later.

All of these cases will be published in detail in a later paper.

Dr. Fred B. Lund, Boston, Mass.:

As Dr. Keen has called attention to the importance of accurate English, it is perhaps as well to be careful to make accurate statements in English. Dr. Bloodgood has just stated that "center" was a good old Anglo-Saxon word. As a matter of fact, "center" is derived from the Latin word "centrum," which comes from the Greek word "kantron," meaning "goad," and, therefore, center is just as good a Greek word as "periphery."

Dr. A. J. Ochsner, Chicago, Ill.:

I wish to direct the attention of the Fellows of this Association to the real virtue of the soldering-iron, which is due to the fact that the large portion of metal will take up a tremendous degree of heat, and that it will hold this heat for a long time. I have used the soldering-iron for thirty-three years in many cases of sarcoma and carcinoma, and I believe that the reason why a large proportion of these cases do not have a recurrence is because of this very great degree of heat that is

carried by this large piece of iron. You apply a piece of metal as large as this with many hundred degrees of heat, and it is sure to carry its heat much further than an ordinary small piece of metal like you have in the Paquelin cautery. We know, from animal experimentation, that if we take a piece of growth, carcinoma particularly, and cut that in half and expose a half of it to a degree of heat of 160° F., and the other half we use fresh, and transplant them both into the same animal, the fresh piece will grow while the other will not, so that here we have a demonstration of the value of heat. In these cases we have always cauterized much more thoroughly than seemed necessary, because slight cauterization is quite useless. We hold the cautery in position for a long time at the same point, so as to be sure that the radiation of heat will reach far beyond the point that we would dare to remove with the knife.

Dr. Howard Lilienthal, New York:

I do not understand Dr. Bloodgood's reason for not making an effort to get a pathological diagnosis in this case. A tiny incision through the skin and the use of Mixter's punch or some similar instrument would suppy a specimen containing enough of the tissue strata for a proper hardening and section. Another matter which I believe of importance is the use of Coley's toxins. I have never lost an opportunity to speak in favor of this agent, which I believe should be made a permanent part of the surgical armamentarium. I have had success in a sufficient number of cases of proven malignancy to make me want to employ the method in almost any case of inoperable sarcoma.

My last patient was a young girl, with a swelling of one ankle, which was diagnosed roentgenologically as a non-malignant bone cyst of the tibia. In these circumstances I operated, finding a soft, solid tumor instead of a cyst. I curetted it out, implanting fat from the patient's abdomen to take its place, and left the posterior part of the tibial wall, which I believed was thick enough to bear her weight. There was primary union. The report of Dr. Mandlebaum was not one to indicate non-malignancy; on the contrary the tumor was a malignant osteosarcoma. With Dr. Codman's figures in mind I did not believe that any local treatment, such as radiation, would be effective. I therefore employed Coley's fluid. The girl later went to San Francisco. Dr. Stillman has kept her under observation and continued to use the toxins. It is now two years since the operation, and the patient is well and is walking. There has been no recurrence.

Dr. E. A. Codman, Boston, Mass.:

I think enough has been said about these bone tumors to illustrate

the fact that what we need is a collection of the evidence. It is up to the individual surgeon to register every case he has and let us have the group to study. The Registry has already collected a large number of cases which are at the disposal of anyone who wishes to write a paper. Dr. Lewis has had some for his paper. For four years I have been reminding surgeons about registering the bone-sarcoma cases, and yet not very many have been registered. There seem to be three reasons for this: First, the fact that we all mean to do it, but we do not, and we forget. Second, there is not time enough; we are busy with other things and we do not have the time to register them. Third, there is the question of the expense of extra records, *x*-ray prints, etc. In order to overcome these difficulties I have invented something which covers all these objections. I am perfectly delighted with it, because in inventing it I have not only invented something to cover all these three points, but something to let me out. I have been tired out writing to my friends reminding them to register their cases, and I shall not have to do this any longer. This little mechanical conscience will do the work for me.

THE REGISTRY OF BONE SARCOMA SCISSORS

This instrument is fully shown in the figures. It consists of the blades of a small pair of scissors with two metal rings welded to the backs of the blades. The metal rings are slipped over the two middle fingers just behind the proximal joints. The surgeon can wear it without hindrance while operating and can cut his ligatures and sutures very readily, and so rapidly that the act appears "sleight-of-hand." Any surgeon can have one made for him by cutting off the ends of a small pair of scissors back of the screw, and having rings, of brass or German silver, welded on the backs of the points. I believe this instrument will be widely used, and although slight details may be changed, the principle of using minute scissors in the clefts between the fingers is likely to survive. It is intended to have a number of these instruments made up and stamped "Registry of Bone Sarcoma." If any surgeon wishes to try one, I will gladly order one for him at cost.

We all want to contribute our bits to surgical technic and to surgical science. This is my bit, for I believe this principle is not in use. So far as I know, it is the first cutting instrument to utilize the power of the interossei and lumbricales. It leaves all the ordinary mechanisms of the hand free for other uses. The idea occurred to me from seeing Lower's ingenious thimble device for the same purpose. If this bit

survives, I hope the idea of the Registry of Bone Sarcoma will survive with it. I would like to see the instrument become a little mechanical conscience, at least as far as registering cases of bone sarcoma is concerned. It will tell its owner that he should do something for medical science in return for the multitude of facts and appliances which medical science daily permits him to use.

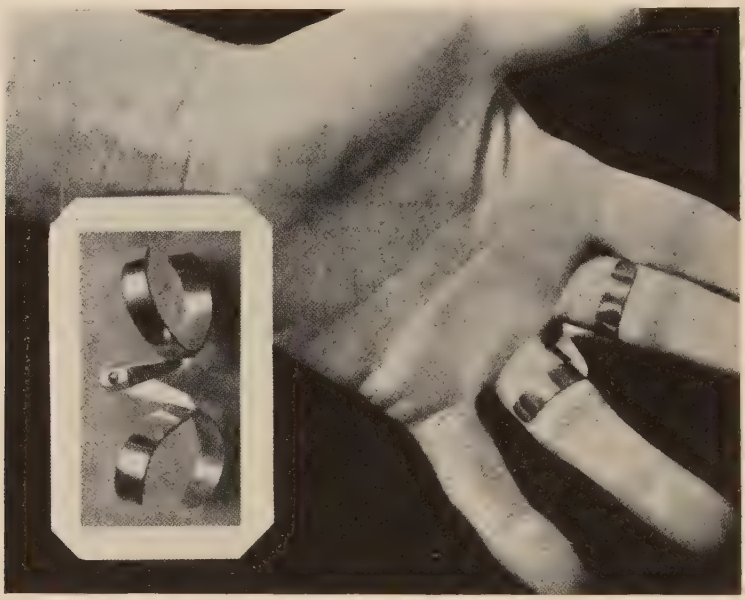


FIG. 1

It is my hope in presenting this instrument to the profession that it will become the fashion to make it socially uncomfortable for any surgeon who is found using this appliance, if he has not recorded his cases of bone sarcoma to the best of his ability. He must be "jollied" into registering or "jollied" out of using the scissors.

Partly to control the name of the instrument and partly to make a fortune out of it, should its use extend to industry and to the household sewing-basket, a patent has been applied for. I am much more likely to make a fortune from this patent than I am from my knowledge of bone sarcoma, for I have repeatedly stated that I shall never make any charges for seeing in consultation patients with this condition. When my ship comes in and all the surgeons in the United States are

reporting their cases, I shall be able to write a beautifully illustrated book on "The Registered Cases of Bone Sarcoma."

A description of the object of the Registry may be found in *Surgery, Gynecology and Obstetrics*, March, 1922, pp. 335–343, and another paper describing its method of procedure is now in press and will soon appear in the same journal, in May, 1924.

I am going to put this instrument on the market, and by the use of it every surgeon can save himself time enough to register every case of bone sarcoma. As to the matter of expense, this can be saved by using a method of suture by which you can get eight stitches out of an ordinary piece of silkworm-gut.

The invention of the "Registry of Bone Sarcoma Scissors," published in the *Boston Medical and Surgical Journal*, April 24, 1924, led to the invention of a new stitch which makes the use of the scissors less necessary.

In making the ordinary interrupted skin suture with silkworm-gut, the surgeon begins with the forceps in his left hand, the points toward the wound, and the needle between his thumb and two forefingers of the right hand. He makes the following motions:

1. He passes the needle through the skin on both sides of the wound (sometimes this is done in two motions).
2. He turns the forceps around in his hand,
3. and ties the first half of a square knot in the end of the suture;
4. then the other half of the knot in the opposite direction.
5. The ends of the suture are then picked up and held taut,
6. and the scissors are picked up and
7. the suture cut.
8. Another motion throws the cut ends away.
9. The forceps are again turned in the hand ready for the next suture.

During this process the assistant may save one or two motions by picking up the scissors and cutting the stitch, but this requires his constant attention, and he might use this time in beginning to clean up the table.

With the new stitch no assistant is needed. Time and suture materal are saved. Six stitches may easily be made with one strand of silkworm-gut instead of two or three. Six stitches with the old method means fifty-four motions besides the rethreading of the needle once or twice. Six stitches by the new method requires only eighteen motions and the positions of the forceps and needle in the hands are practically unchanged. Scissors need not be used.

Starting in the same position: (Fig. 2) Pass the needle through the

lips of the wound from right to left. (Fig. 3) The needle is passed under the end of the suture and drawn through to the right, thus forming the first half of the square knot. (Fig. 4) The needle is then passed over the forceps and under the end of the suture to the left, thus completing the first stitch.

The suture is not cut, but a short loop is left as shown in Fig. 5, and a new stitch started with the fourth motion, which is the first motion of the second stitch. The positions in Fig. 5 and Fig. 3, therefore correspond.

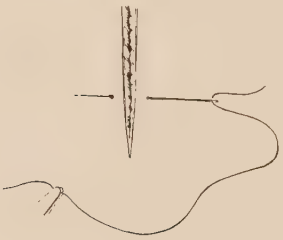


FIG. 2

FIG. 3

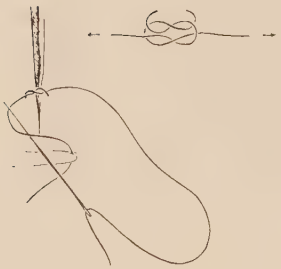


FIG. 4

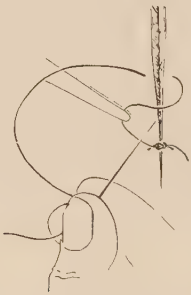


FIG. 5

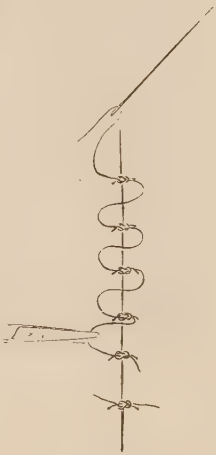


FIG. 6

No cutting of the suture need be done, the one suture having made the six stitches as shown in Fig. 6. The lower suture is shown as cut, but this is unnecessary, for the dressing can be applied without cutting any of the loops. There will be only two instead of twelve ends to occasionally stick into the flesh and annoy the patient. When it is time to take them out you will not have to hunt for the ends. Each knot can be cut and all removed together.

The same kind of stitch can be used for deep sutures when a needle holder is used, but two motions will be added for each stitch as the

needle is grasped after passing through each knot. The needle holder cannot jump around the stitch as the surgeon's fingers do when he rolls the pressure of his thumb from his second to his forefinger, imperceptibly crossing the suture with his thumb without dropping the needle. There will also be the extra motions of cutting the intermediate loops. Even at that it is much more economical than other sutures.

I think it is conservative to say that 50 per cent of suture material can be saved by this stitch.

I had the opportunity of presenting this method, with the "Registry of Bone Sarcoma Scissors," at the meeting of the *American Surgical Association* at Baltimore on April 18, 1924. None of those I showed it to recalled having seen the suture before, except Dr. Bloodgood, who said that he had seen it in Germany when he was a student, silver wire being used.

The Ochsner Clinic has used the best part of it, *i. e.*, tying the knot with the needle, for many years. But they have not used the series of sutures without interrupting, having stopped to cut off each individual stitch. Omitting cutting between each stitch saves still more time and suture material.

I have given the above name to the suture, for the same reason that the scissors were named, *i. e.*, to recall to each surgeon who uses it that he, too, has an opportunity to contribute his bit to surgical science by faithfully reporting every case of bone sarcoma which comes to his notice.

By saving assistants' time and suture material, the idea may also serve to compensate in the minds of hospital superintendents the expense of duplicate *x*-rays, histories and miscroscopical slides which are sent to the Registry.

I make the same request to those who use this stitch as I did to those who may use the scissors—that they link the name of the Registry with it. Perhaps the stitch may have been described in by-gone text-books, but if so it must have been forgotten or we should see it in use in this hurried generation. Surgeons who save time with this stitch and hospitals which save suture material with it can render detailed reports of their bone sarcoma cases and still save time and money.

THE EXPERIMENTAL TRANSPLANTATION OF THE DIAPHRAGM AS AN ADJUNCT IN THE TREATMENT OF LESIONS AT THE LOWER END OF THE ŒSOPHAGUS

THE APPLICATION OF THE METHOD IN A CASE OF CARCINOMA OF THE CARDIAC END OF THE ŒSOPHAGUS IN MAN

By GEORGE J. HEUER, M.D., WILLIAM De W. ANDRUS, M.D.
(By Invitation.)

AND

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CINCINNATI, OHIO.

The problem of resecting a tumor of the œsophagus and reëstablishing the canal has interested surgeons for many years—indeed, since about 1885. A truly enormous amount of experimental and clinical work has been done in an attempt to solve the problem, but as yet with very meager results. In the whole history of œsophageal surgery a malignant tumor has been successfully removed from the thoracic œsophagus in the human being but 6 times,[1] and in only 1 of these was the canal reëstablished. The difficulties have been chiefly five: (1) The condition of the patient, which is generally poor; (2) the preservation of the blood supply of that part of the œsophagus which has been mobilized for resection; (3) a secure suture either between the ends of the divided œsophagus or between the œsophagus and some other viscus; (4) the bridging of a gap after the resection of a portion of the œsophagus; and (5) the prevention of a fatal

[1] Since this was written, Eggers, of New York, has reported an additional successful case.

intrapleural infection. It will not be necessary to go fully into these difficulties, for they are perfectly familiar to surgeons. They have, however, been so formidable that they have compelled surgeons to devise long, tedious and intricate procedures, either extrapleural or extrathoracic, for use in operations upon man. These have required a period of months to complete, and the patients subjected to them have usually died before they have been completed. Meanwhile, surgeons have continued in their efforts to find a simple and satisfactory intrathoracic method which might safely supplant the extrapleural or extrathoracic methods.

The simplest experimental intrathoracic method was first suggested by Biondi in 1896. He freed the œsophagus at the hiatus, drew the stomach through the diaphragm into the thorax, resected the œsophagus and anastomosed the upper end of the divided œsophagus with the stomach. The same idea with various modifications formed the basis of experimental work by Anschutz, Sauerbruch, Willy Meyer, Green and Janeway, Zaaijer, Omi, Enderlen, and Miller and Andrus. The method has been reasonably successful in dogs, but there have been the complications due to imperfect suture with infection, of dilatation of the transposed stomach and of diaphragmatic hernia. It has never been successfully carried out in man.

Drs. Andrus, Bell and I thought a wholly new approach to the problem might contribute something toward its solution. Instead of attempting an anastomosis between œsophagus and stomach within the thorax, we considered transferring the lower one-third of the œsophagus into the abdomen and performing the anastomosis there. The obvious advantages of such a procedure are that it permits a primary gastrostomy for feeding purposes; it avoids the transposition of stomach or intestine into the thorax, which is not well borne, and permits an anastomosis in the abdomen where infection, if it should occur, is less fatal than in the thorax. In order to convert the lower end of the œsophagus into an abdominal viscus, one must transplant the diaphragm upward; and having in mind a two-step procedure, our work

naturally was divided into two parts: (I) To test the feasibility, safety and possible complications of transplantation of the diaphragm, and (II) to perform a successful intraäbdominal œsophagogastrostomy after resection of the œsophagus.

I. The Transplantation Upward of the Diaphragm. (*A*) Procedure 1. Under intratracheal anesthesia a left intercostal incision is made over the 9th rib (which corresponds to the 8th rib in the human) and the thorax opened widely. The left half of the diaphragm is seized and incised close to and parallel with the thoracic wall, so as to free the entire half of the diaphragm, including the crux, from its attachment. The œsophagus is not freed from the diaphragmatic hiatus. The cut edge of the mobilized diaphragm is then brought upward four or five interspaces and sutured to the pleura, the bite of the needle including the intercostal muscles. The thoracic wound is closed. The object of this procedure was to test the technical difficulties and the possible complications of this simple operation.

Results. This procedure, combined with division of the phrenic nerve, was carried out upon 7 dogs. The only technical difficulty—and this is not great—is the division of the diaphragm near the left crux, where an artery of considerable size may give rise to annoying bleeding. The animals recovered promptly from the operation, were active and ate and slept well. They were never observed to vomit their food. Although the left lung was in part compressed by the elevation of the diaphragm, they did not show any respiratory embarrassment. Of the 7 animals, 1 died upon the fourth day after operation from an infection and 1 was killed in a fight upon the twelfth day. The remaining dogs were sacrificed upon the fifth, tenth, twelfth, sixty-eighth and seventy-eighth days after operation. In all 7 animals the transplanted diaphragm was found firmly attached to the thoracic wall. Its musculature, due to the deliberate division of the phrenic nerve, had become, in the animals sacrificed late, markedly atrophic. There was no instance of diaphragmatic hernia.

(*B*) Procedure 2. Under intratracheal anesthesia a left

intercostal incision is made over the 9th rib and the thorax opened widely. The left half of the diaphragm is seized and incised close to and parallel with the thoracic wall, so as to free the entire half of the diaphragm, including the crux, from its attachment (Fig. 1). The œsophagus is freed from the hiatus and isolated almost to the bifurcation of the trachea. The dia-

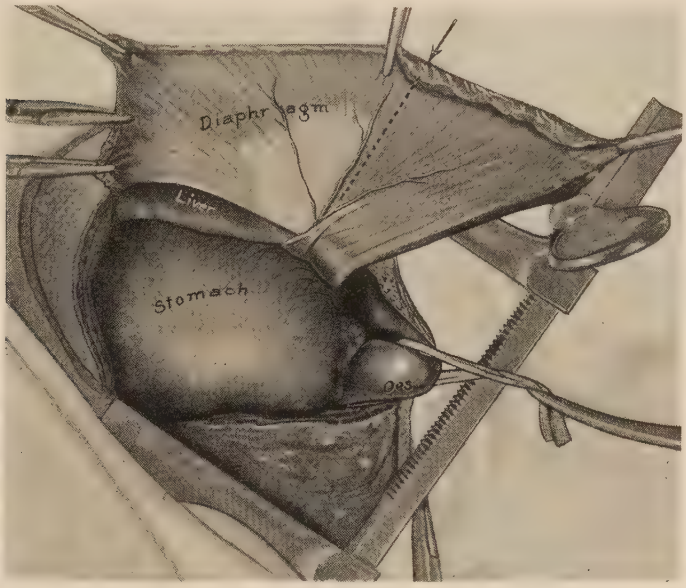

FIG. 1.—Transplantation upward of the diaphragm. The pleural cavity has been opened widely. The entire left half of the diaphragm including the crux has been incised close to its attachment to the thoracic wall and brought upward. The œsophagus has been freed from the diaphragm at the hiatus. The dotted line on the mobilized diaphragm indicates the position of the incision used in the formation of the new hiatus.

phragm is split into its tendinous portion, so as to allow the formation of a new hiatus and the split resutured around the œsophagus (Fig. 2). The cut edge of the diaphragm is then brought upward four or five interspaces and sutured to the pleura, the bite of the needle including the intercostal muscles Figs. 3 and 4). The result is a marked elevation of the diaphragm

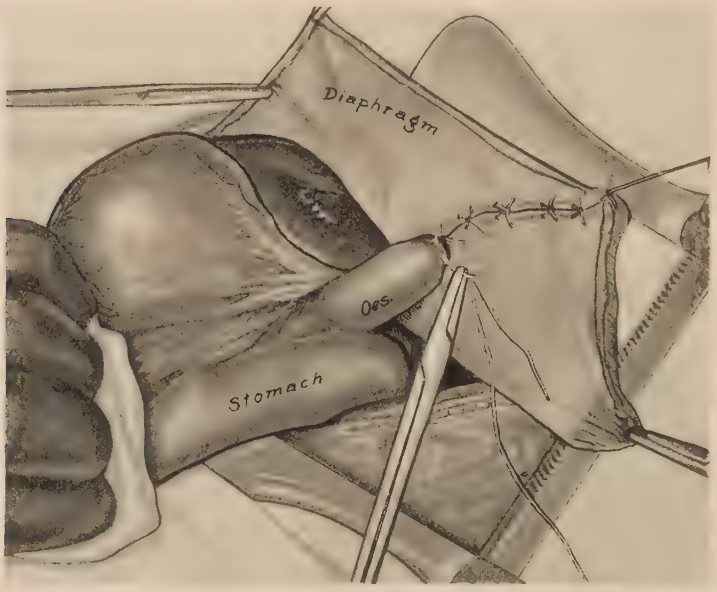

FIG. 2.—The formation of the new hiatus completed.

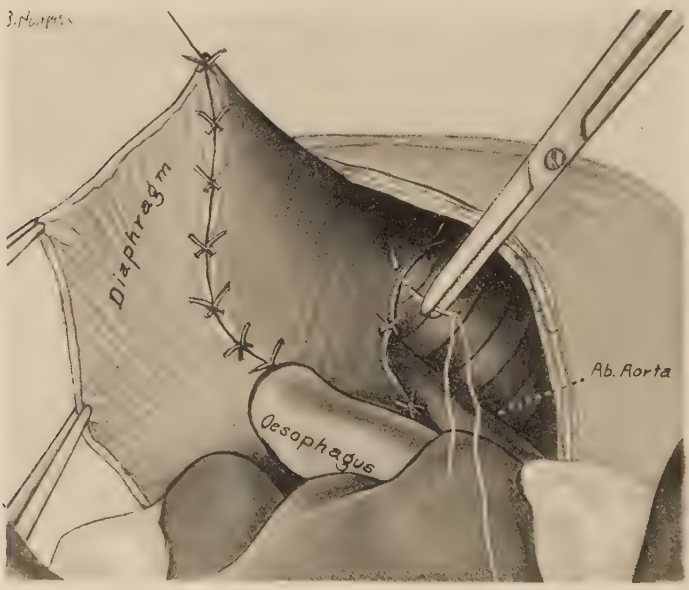

FIG. 3.—The suture of the cut edge of the diaphragm to the thoracic wall four to five interspaces above its original position.

and the conversion of the lower one-third or more of the œsophagus into an abdominal viscus (Fig. 5). The thoracic wound is closed. The phrenic nerve was not divided.

Results. This procedure was carried out upon 19 dogs. The operation presents but two technical difficulties: The division of the diaphragm near the left crux, as noted under Procedure 1, and the satisfactory suture of the transplanted diaphragm to the posterior thoracic wall at the spine and around the aorta. It is here that the liability to the development of diaphragmatic hernia is greatest because of the absence of firm tissues to support

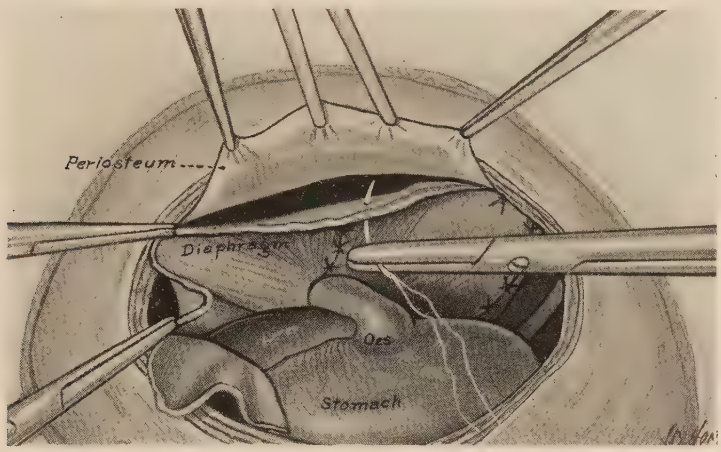

FIG. 4.—The transplantation of the diaphragm nearing completion.

a suture. The isolation of the œsophagus in the dog is very easy and can be accomplished without opening the opposite pleural cavity.

The animals operated upon recovered promptly from the procedure and failed to show respiratory embarrassment or other untoward symptoms. Four of the animals were repeatedly fluoroscoped from forty-eight hours to eighteen days after operation and showed that the transplanted half of the diaphragm moved on inspiration and expiration, although its excursions were less than those of the normal side. Repeated observation at autopsy

FIG. 5.—Autopsy specimen showing the lower portion of that part of the œsophagus which has been transposed into the abdomen. The thread is attached to the costal margin. The transplanted diaphragm is above this level and does not show in the photograph.

showed that the musculature of the transplanted diaphragm was normal in appearance and thickness.

COMPLICATIONS. 1. *Accidental.* Seven of the animals developed either a wound or an intrapleural infection, due to faulty aseptic technic. Three animals died on the tenth, twenty-second and sixty-third days after operation, and at autopsy were found to have strangulated, one from a hair-ball and two from bones. The point of lodgment of the foreign bodies was in the œsophagus at the new œsophageal hiatus. Two animals were found dead on the eleventh and thirty-sixth days after operation, and at autopsy failed to show any lesions which could account for their deaths.

2. *As a Result of the Operation.* (*a*) *Diaphragmatic Hernia.* In 2 animals sacrificed several months after operation a single suture uniting the diaphragm with the posterior thoracic wall was found to have pulled away, allowing a small opening between the abdominal and thoracic cavities in one instance and between the abdominal cavity and posterior mediastinum in the other. In neither case had a diaphragmatic hernia resulted. In 1 animal death occurred on the thirty-sixth day after operation and at autopsy a diaphragmatic hernia involving the stomach and spleen was found. This is the only instance of diaphragmatic hernia in the series. Why in this animal the diaphragm had in part pulled away from the thoracic wall could not be explained. In no instance was the diaphragm sutured to the thoracic wall under tension.

(*b*) *Vomiting or Regurgitation.* The most constant complication of Procedure 2 was the occurrence of vomiting. In 15 of the 19 animals more or less vomiting, with a tendency to lose weight, occurred. In 4 animals both vomiting and loss of weight were completely absent. The vomiting was distinctly of the œsophageal type, a regurgitation of food immediately after its ingestion. Autopsies upon such animals several weeks or months after operation showed a slight narrowing of the œsophagus at the new hiatus, this narrowing apparently being due to the contraction of the diaphragm around the œsophageal wall. There

was hypertrophy of the wall of the œsophagus above the point of constriction and, in a few instances, slight dilatation as compared with the œsophagus below the hiatus. No dilatation comparable to that seen in cardiospasm or cardiac obstruction in man was observed. In the 4 animals which did not vomit it was noted that the new hiatus was more loosely formed around the œsophagus. It is quite possible that our fear of diaphragmatic hernia was exaggerated and that we fitted the diaphragm too snugly around the œsophagus. The problem of forming a new hiatus sufficiently wide to prevent the occurrence of vomiting or the development of diaphragmatic hernia could, we think, be solved through the use of a cuff of fascia.

(*C*) Procedure 3. This procedure differs from the preceding in that (1) the esophagus is not dissected from its bed, but is simply freed from the diaphragm at the hiatus, and (2) the mobilized diaphragm is not split to form a new hiatus, but is brought upward to the desired level and sutured to the posterior thoracic wall around the œsophagus and aorta. The considerations which led to these modifications of Procedure 2 were the occurrence of gangrene of the œsophagus in a case of carcinoma, to be detailed later, and due, in our opinion, to the extensive mobilization in the first stage of the operation; and the occurrence of vomiting in the animals subjected to Procedure 2, which we thought to be caused by constriction of the œsophagus at the newly formed hiatus. The procedure technically is simpler in some respects than Procedure 2, but is more difficult, in that a secure suture of the diaphragm to the posterior thoracic wall around the œsophagus and aorta is less easily made.

Results. Only 2 dogs were subjected to this procedure, too small a number from which to draw conclusions. A later report on this phase of the work will be forthcoming. Both animals recovered promptly from the operation and were never observed to vomit or regurgitate their food. They maintained their weight. They were sacrificed on the fifty-fourth and seventieth days after operation. One was found to have a diaphragmatic hernia involving portions of the stomach, small intestine and spleen. In the other the result was perfect.

II. Intraäbdominal Œsophagogastrostomy. In the contemplated resection of the lower end of the œsophagus, Procedure 2, or, should it prove more satisfactory, Procedure 3, constitutes the first stage of the operation. The second stage consists of the resection of the œsophagus and the anastomosis between the cephalic end of the divided œsophagus and the fundus of the stomach. After a number of experiments the technic of œsophagogastrostomy, as described by Miller and Andrus,[1] but performed within the abdomen instead of within the thorax, was adopted as being most satisfactory.

Procedure. In animals previously subjected to Procedure 2 or 3 an intercostal incision is made an interspace below the previous incision, and the abdominal cavity opened widely. The œsophagus, previously transferred into the abdominal cavity by the elevation of the diaphragm, is resected between ligatures and the divided stumps cauterized. The distal stump is inverted into the stomach. The proximal stump is anastomosed with the fundus of the stomach. (See Figs. 6, 7, 8, 9, 10 from Miller and Andrus's paper.[2]) It is quite easy to resect the lesser curvature of the stomach with the lower end of the œsophagus through this approach. The intercostal incision is closed.

Result. The procedure technically is very easy. The approach to the cardiac end of the stomach is short and direct. The fundus of the stomach can easily be brought up to the level of the transplanted diaphragm without tension and without the division of any of its vessels. The placement of the posterior sutures between the œsophagus and fundus of the stomach naturally constitutes the most difficult part of the procedure (Fig. 11).

Six animals subjected to Procedure 2 or 3, from fourteen to one hundred and twenty-five days previously, were again operated upon and an œsophagogastrostomy performed. One animal died upon the operating table just after the complete closure of the wound. Immediate autopsy showed a marked mediastinal

[1] Johns Hopkins Hospital Bulletin, April, 1923, p. 109.

[2] The authors wish to gratefully acknowledge the loan of the cuts for Figs. 6 to 10 by the Johns Hopkins Hospital Bulletin.

FIG. 6.—The stomach drawn into the wound, showing the site of anastomosis with the œsophagus. In our procedure the vasa brevia are not ligated as shown in the illustration.

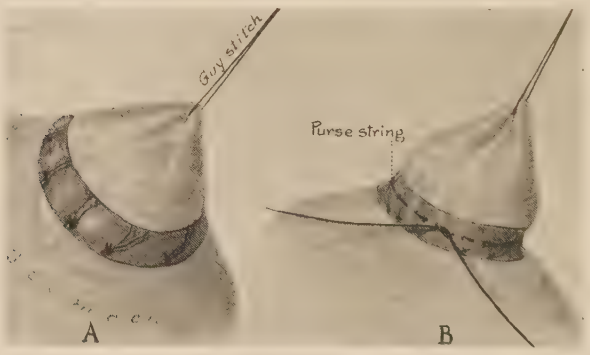

FIG. 7.—Preparation of the site for implantation in the stomach wall.

emphysema perhaps sufficient to have embarrassed the heart. It is the only instance of the occurrence of this complication, and no doubt was due to insufficient care in our manipulations.

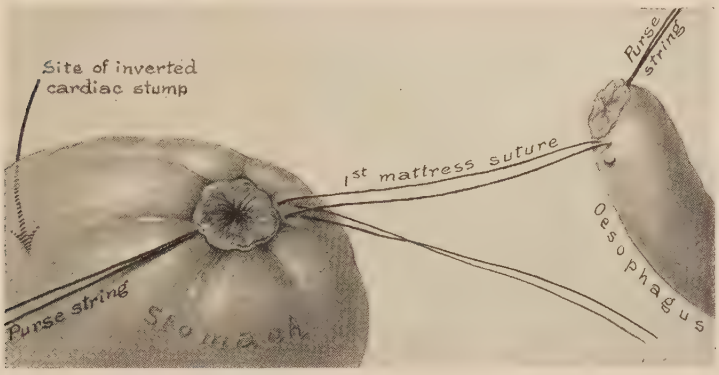

FIG. 8.—Beginning of the stitch anastomosing the œsophagus to the stomach.

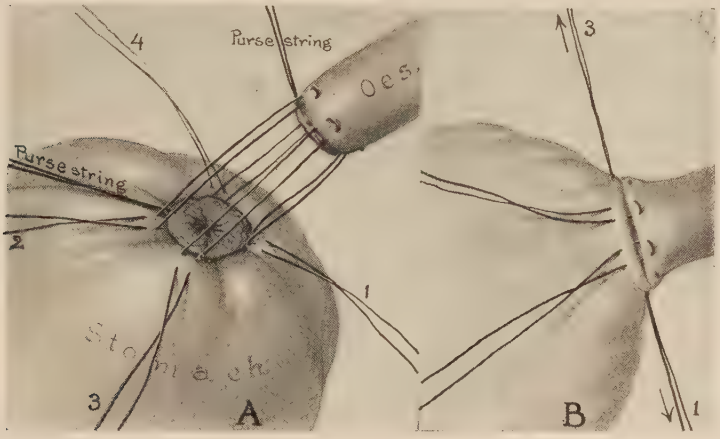

FIG. 9A.—The four cardinal mattress stitches have been placed.

FIG. 9B.—The first layer is complete except for tying the two final sutures.

The suture line was water-tight. In 4 animals the suture between œsophagus and stomach was perfectly satisfactory and there was no leakage from the suture line. These animals made

perfect operative recoveries. One animal developed signs of peritonitis and died upon the seventh day. At autopsy leakage was found to have occurred from an imperfect suture line with the development of a purulent peritonitis.

Of the 4 animals which recovered, one is living and in good health four months after operation. Two animals which vomited following the first stage (Procedure 2) stopped vomiting for six

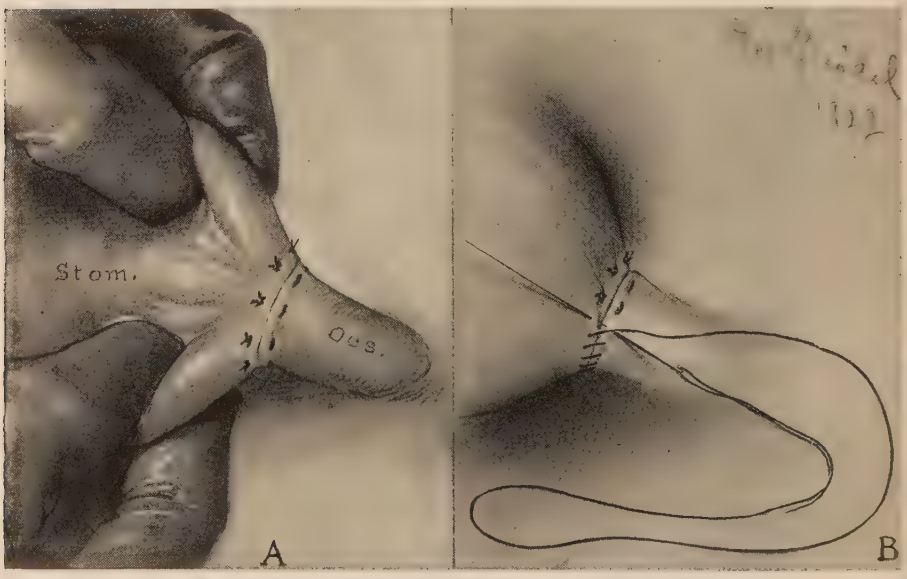

FIG. 10A.—The first layer completed.

FIG. 10B.—Placing of the second and final suture layer.

to eight days after the œsophagogastrostomy, but again began to vomit. At autopsy on the forty-fifth and forty-sixth days after the œsophagogastrostomy they presented, as previously noted, constrictions of the œsophagus at the new hiatus, but in addition had strictured openings at the site of anastomosis between œsophagus and stomach (Fig. 12). The remaining animal has recently been sacrificed, and at autopsy showed a perfect result.

SUMMARY AND CONCLUSIONS. 1. A two-stage operation for the resection within the abdomen of the lower end of the œsoph-

FIG. 11.—Autopsy specimen showing completed œsophagogastrostomy with the diaphragm attached to the œsophagus, some distance above the site of anastomosis.

agus and the cardiac end of the stomach is suggested. The first stage contemplates the transposition of the lower end of the œsophagus into the abdomen through the transplantation upward

of the left half of the diaphragm. The second stage includes the resection of the œsophagus and an œsophagogastrostomy. Whether the freeing of the œsophagus from its bed should be included in the first stage or left until the second stage, has not, in our own minds, been definitely determined. Our experience

FIG. 12.—Specimen showing strictured opening of the site of anastomosis between œsophagus and stomach. The hypertrophy of the œsophageal wall is evident.

in one human case in which gangrene of the œsophagus, isolated during the first stage, occurred, suggests strongly that this step be left until the second stage. A gastrostomy preliminary to the first stage may be done without interfering with the technical steps of this procedure.

2. The transplantation upward of the left half of the diaphragm is a simple and reasonably safe procedure. It has been done 28 times in dogs and once in the human being. The occurrence of diaphragmatic hernia in 2 instances in dogs suggests that this is the complication most likely to occur. Sufficient care in the placement of the sutures should avoid this complication. Whether the phrenic nerve to the transplanted diaphragm be divided or not, does not, in our experience, make any difference in the result. In the 1 instance the diaphragm remains immobile, occupies a high position, and eventually, through atrophy, is converted into a fibrous sheet. In the other instance the transplanted diaphragm moves normally, but less in amplitude, and its musculture remains intact. It occurred to us that a mobile diaphragm by possible traction on the œsophagus might interfere with its function.

3. The formation of a new œsophageal hiatus which will not constrict the œsophagus, and at the same time not permit the development of diaphragmatic hernia, has proven to be the most difficult problem in connection with transplantation of the diaphragm. Our experience with Procedure 2 suggests that the frequent occurrence of vomiting was due either to the constriction of the œsophagus by the diaphragm or traction upon this structure during movements of the diaphragm. Procedure 3, in which neither factor is operative, would appear the better and will be given a further trial. In the 2 animals subjected to it vomiting did not occur nor was there at autopsy any constriction of the œsophagus. It should be noted, however, that vomiting has frequently been observed in the experimental work of others in which constriction of or traction upon the œsophagus could not very well have occurred.

4. Oesophagogastrostomy performed within the abdomen through an intercostal incision is technically not difficult. The operation can be performed without mobilization of the stomach. Resection of the cardia and lesser curvature of the stomach is quite feasible. The experience of previous investigators has shown that an end-to-end, lateral or end-to-side anastomosis

between œsophagus and stomach when performed within the thorax is, in the dog, a reasonably safe procedure—so safe that it may be justifiably transferred to the human being. Our experience confirms this view. Performed within the abdomen where omentum and other structures are available to protect the suture line it should be safer still.

The application of the above method in a case of carcinoma of the lower end of the œsophagus:

Case Report. F. G., aged sixty-one years, was admitted to the Cincinnati General Hospital January 24, 1924, complaining of difficulty in swallowing, vomiting and pain in the pit of the stomach. Previous to November, 1923, he had excellent health. About Thanksgiving Day of that year he began to have pain in the lower lateral portion of his right chest. This came on suddenly, was continuous and was at first confined to an area the size of a silver dollar. Later he had pain behind the lower end of his sternum, which was distinctly worse after taking food. He visited a physician, who after *x*-ray studies told him he had a constriction at the lower end of the œsophagus. About January 1, 1924, he began to have difficulty in swallowing solid or semisolid food, the ingestion of which was almost immediately followed by its regurgitation. Since that time he has lived on fluids which he can swallow satisfactorily. He has lost twenty pounds in weight. He entered the hospital for the relief of the obstructive symptoms.

Physical Examination. A well-developed, although poorly nourished, rather cachectic-looking white man who evidently had lost considerable weight. The examination of the head, neck, thorax, abdomen and extremities showed nothing remarkable. There was no enlargement of the supraclavicular glands and no masses palpable in the abdomen. The Wassermann reaction was negative. Fluroscopic studies of the œsophagus by Dr. Little showed an incomplete obstruction about 2 inches above the cardiac orifice of the stomach. Œsophagoscopic examination by Dr. Iglauer showed a nodular growth on the

anterior wall of the lower portion of the œsophagus, 15.5 inches from the incisor teeth. It was diagnosed a carcinoma.

Operation. (Dr. Heuer, February 14, 1924.) Exploration of a carcinoma of the lower end of the œsophagus. Freeing of the lower 11 cm. of the œsophagus from its bed. Transplantation of the left half of the diaphragm, transferring the lower portion of the œsophagus, including the tumor, into the abdomen. Closure. Intratracheal anæsthesia (Dr. Andrus).

With the patient on his right side a long incision was made over the 8th rib and perhaps twelve inches of this rib resected. The pleura was opened widely and the ribs separated with a rib-spreader. The lung collapsed immediately and there were no adhesions. Examination immediately revealed a tumor of the œsophagus, the lower end of which began just above the œsophageal hiatus and extended $7\frac{1}{2}$ cm. upward. The tumor was $5\frac{1}{2}$ cm. in diameter. With our dog experiments in mind we began to free this tumor from the surrounding structures. In front, it was densely adherent to the pericardium, but separation from this structure was not especially difficult, although we had to stop periodically because of the irregularities in the heart caused by our manipulation. Lifting the tumor out of its posterior bed was a much more difficult matter. The tumor was found adherent to the wall of the aorta, and great care was exercised in separating it from this structure. In the course of the separation two large arteries, almost the size of a radial, were found running from the artery directly into the region of the tumor. These had to be doubly ligated and divided. As a result of this procedure, the tumor mass assumed a bluish discoloration, but I thought that the circulation in the portion of the œsophagus which I had freed was perhaps adequate. After a considerable period of time we were able to free the entire tumor from the surrounding structures and to isolate the œsophagus above the upper limit of the tumor for about two inches. We therefore completely freed the œsophagus from its bed over a distance of about 11 cm. After getting this far, the patient's condition still being satisfactory, we incised the left half of the

diaphragm close to its attachment to the thoracic wall and freed the œsophagus from the diaphragm at the hiatus. We then split the mobilized left diaphragm so as to form a new hiatus and resutured the split around the œsophagus. The diaphragm was then moved upward to a point two inches above the upper margin of the tumor and sutured to the thoracic wall around its entire hemicircumference. Two sutures were placed, fixing the diaphragm to the œsophageal wall at its transplanted point. At the completion of the procedure we had converted the lower 11 cm. of the œsophagus, including the tumor, into an abdominal viscus, and we proposed at a second stage to resect the tumor and make an anastomosis between the fundus of the stomach and the upper end of the divided œsophagus. The wound was closed with silk without drainage.

Pathology at the Operating Table. The tumor, as noted, measured 7.5 cm. x 5.5. cm. in diameter. It was very hard on palpation and was adherent both to the pericardium in front and the aorta laterally. It could, however, be cleanly separated from these structures. Palpation of the mediastinum after freeing the esophagus failed to show any enlarged mediastinal glands. One or two small glands adherent to the tumor were freed with it and subsequently removed. Palpation of the stomach showed a chain of enlarged glands along the lesser curvature which it was planned to remove at the second stage. Our examination, therefore, would indicate that this was an operable tumor.

Subsequent History. The patient withstood the operation very satisfactorily. For forty-eight hours he was in good shape, then he developed fever, sank rapidly and died.

Autopsy. Upon opening the thorax there was a small amount of purulent fluid in the left lower thorax. In the upper abdomen about the tumor mass there was also a small amount of purulent fluid. Examination of the œsophagus showed an area of gangrene involving the proximal half of the growth and extending upward upon the uninvolved œsophagus to a point 0.5 to 1.0 cm. above the new diaphragmatic hiatus. At one point on the right anterolateral wall a perforation had occurred in the gangrenous area, allowing leakage into both pleural and abdominal cavities.

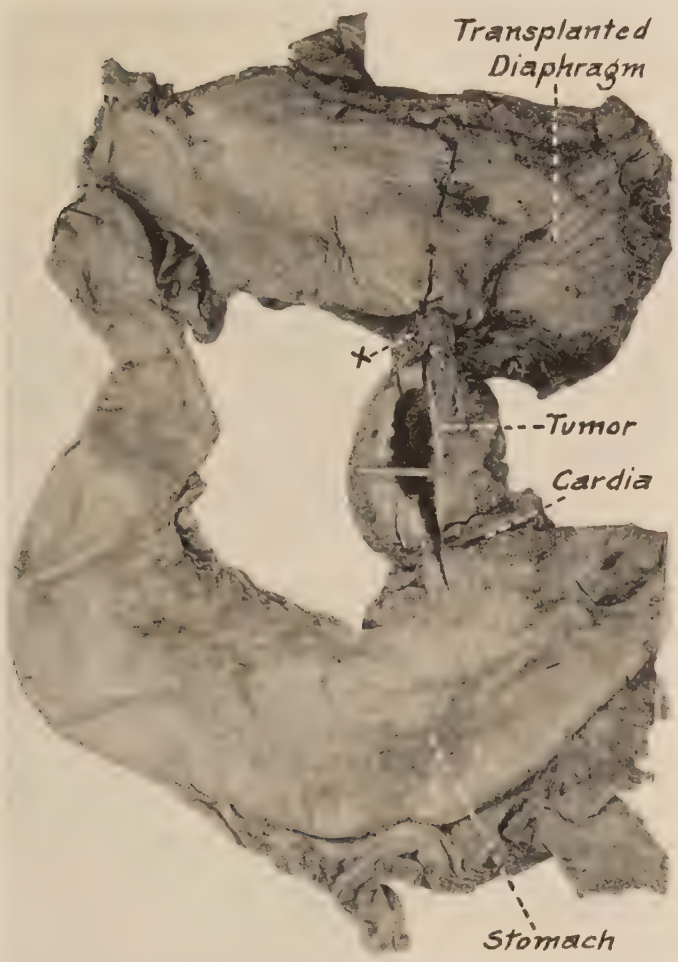

FIG. 13.—Human specimen of carcinoma of the lower end of the œsophagus, showing relations of stomach, cardia, tumor and transplanted diaphragm. The specimen has shrunken, due to fixation, and gives no idea of the segment of œsophagus between the upper margin of the tumor and the diaphragm. This at the completion of the first stage was 2 inches in length, sufficient for a subsequent œsophagogastrostomy. × indicates the site of perforation of the gangrenous œsophagus.

The suture of the diaphragm to the thoracic wall was everywhere perfect. None of the sutures had pulled away. There was no diaphragmatic hernia. Death evidently was due to

FIG. 14.—Posterior view of human specimen. The new hiatus is well seen.

infection secondary to gangrene, with perforation of the œsophagus. The specimen showing the relations of stomach, cardia, tumor and transplanted diaphragm is shown in Figs. 13 and 14. A thorough examination of the body failed to show any involved

glands other than those attached to the tumor and those along the lesser curvature of the stomach.

Pathological Diagnosis. Carcinoma of the œsophagus, squamous cell.

Comments. The first stage of the operation was, with the exception of freeing the tumor from the pericardium and aorta, quite as easy as in the dog. Our great mistake in this case was the freeing of the œsophagus in the first stage. During this procedure the ligature and division of two good-sized vessels extending from the aorta to the œsophagus was followed by visible impairment in the circulation of the œsophagus; and we should either have deferred the mobilization of the œsophagus until the second stage or attempted to complete the entire operation at one sitting. Had we done the latter, however, it is doubtful that the patient would have survived it, and we would have obviated one of the advantages which the two-stage operation was intended to give, *i. e.*, the sealing off of the pleural cavity before the performance of œsophagogastrostomy. Our opinion at present is that the first stage should include only the transplantation upward of the diaphragm, and that the freeing of the œsophagus should be included with œsophagogastrostomy in the second stage.

DISCUSSION

Dr. Robert T. Miller, Jr., Baltimore, Md.:

Dr. Heuer has offered a very interesting suggestion in his paper. The operative proposal which he has made is limited in its application and concerns only the lower third of the œsophagus; but it is in the management of tumors in this situation that we are most likely to make advances. The lower third of the œsophagus is relatively free, and for this reason the difficulties encountered are somewhat simpler than those encountered at the higher level. I have seen his operation carried out in the dog once, and am very glad to say that the suggestion seemed quite practical. With reference to infection, more particularly that resulting from the suture itself, transplantation of the diaphragm offers an excellent chance of converting a dangerous thoracic complication into a simpler abdominal complication. It is to be said that an anastomosis of the œsophagus and the stomach carried out after the plan shown in the pictures can be practised in the animal with a minimal danger of infection, and there seems reason to believe that it is more important to obtain a safe method of suture rather than to devise means of managing an infection due to an insecure stitch. It is, unfortunately, true, however, that any stitch of the œsophagus at present carries with it an element of uncertainty, and for this reason transplantation of the diaphragm by the procedure suggested is a welcome addition to our operative methods. Resection of the lower third of the œsophagus in the dog with restoration of the continuity of the digestive tract is sometimes followed by dilatation of the stomach. This complication, while not particularly frequent, is, nevertheless, apt to prove fatal in the dog by reason of the great degree of pressure exerted upon the heart and lungs by the distended stomach. Practically everyone who has done any experimental work in this field has encountered this difficulty. There is reason to believe that it is the result of destroying the vagus supply to the stomach; it is impossible to resect satisfactorily a growth in the region of the cardia, and probably in any portion of the lower third of the œsophagus as well, without resecting both vagus nerves. If the stomach be transplanted to the chest after the proposal I made in 1923, it inevitably brings about pressure upon heart and lungs. If, however, the diaphragm were transplanted the stomach would still remain an abdominal organ, and it is entirely possible that distention of the stomach would not prove so dangerous. In the work which Dr. Andrus and I did we each of us received the impression that the

stomach after a time regains its tone and that dilatation of the stomach as a serious complication is therefore limited in its occurrence to a relatively brief postoperative period. It follows, therefore, that if some means can be devised for safeguarding the patient during this transient period the danger from this source will be eliminated, and the proposal Dr. Heuer makes seems to offer definite improvement in regard to this point.

I have had one opportunity to attempt the resection of a cancer of the lower third of the œsophagus in the human. We used the transthoracic route of approach and were able to free the growth and resect it. The lower end of the œsophagus was then anastomosed to the fundus of the stomach without great difficulty. The operation, however, caused quite a marked degree of shock, and the patient died at the end of twenty-four hours. Unfortunately, autopsy was refused, so that the help to be obtained from this case is limited to the impression we received during the operation. Those of us concerned in this case gained a very distinct impression that the transthoracic operation is a thoroughly practical procedure, and there seemed to each of us every reason to believe that with opportunity the method could be developed to great usefulness.

Dr. Heuer's paper emphasizes one other thing, namely, that the surgical problem offered by tumors of the œsophagus differs greatly according to the level at which the tumors occur. In the present state of development of this branch of surgery, operation for removal of cancers of the upper thoracic œsophagus is almost forbidden by reason of the difficulty of approach and the complication offered by traumatization of the vagus nerve at this high level, and finally the extraordinary difficulty of restoration of continuity of the tube. For this reason we have felt that for the present it is well to confine our operative work to tumors of the lower third, and all cases seen in the last two years have been reviewed with this in mind. Unfortunately the rule is so stringent as to have excluded from operation all of the cases we have seen. If the difficulties can be met and successfully surmounted in the lower third, where they are in their simplest form, it must follow that operative attack on the rest of the thoracic œsophagus will result. Dr. Heuer is to be congratulated upon having made a very practical suggestion.

Dr. W. W. Keen, Philadelphia, Pa.:

I hardly know what to compliment most, the ingenuity or the audacity of Dr. Heuer. As I sat listening to his paper, I have been wondering if I could double the length of my life, which would carry me over to 2011, what the surgeons will be doing then!

Dr. Willy Meyer, New York:

Dr. Heuer's work is splendid and I do hope he will continue it. For the resection of the cardia the transplantation of the diaphragm must be a very great advantage when making an anastomosis between the lower end of the œsophagus and the stomach by placing the operating field intra-abdominally and leaving the entire upper portion of the normal œsophagus undisturbed. For cancer of the upper portion of the œsophagus, about six or eight inches above the cardia, I think transpleural resection with transplantation of the proximal stump under the skin of the chest and then closing the thoracic wound will likely become the operation of choice. Torek's case, operated on eleven years ago, proves this. That patient is entirely well today and swallows everything through a rubber tube which connects the antethoracic œsophageal opening with that of the gastrostomy. The present condition of the patient is going to be reported by Dr. Torek at the coming meeting of the American Association for Thoracic Surgery.

PRIMARY GIANT-CELL TUMORS OF THE VERTEBRÆ

ANALYSIS OF A GROUP OF CASES, WITH REPORT OF CASE IN WHICH PATIENT IS WELL TWO YEARS AND NINE MONTHS AFTER OPERATION

By DEAN LEWIS, M.D.
CHICAGO, ILL.

Tumors developing from the vertebral column are more common than those developing from the meninges or cord. The metastatic outnumber the primary growths, as is indicated by the figures published in 1898 relating to 107 tumors of the vertebræ: Carcinoma, 54 cases; sarcoma, 34; osteoma, 10; myeloma, 6; chondroma (chondrosarcoma), 2; myxoma, 1.

It is difficult to determine the frequency with which giant-cell tumors occur in the vertebræ. It has been suggested that they may be mistaken for tuberculosis. The few case reports that have been published would seem to indicate that such tumors are uncommon.

Bloodgood, in his latest communication dealing with giant-cell tumors, reported 77 cases. Since this report appeared, almost a year ago now, Codman has sent him the registered cases; so that these, with the new cases which he has, total 179. Five of these tumors occurred in the vertebræ.

Report of the Author's Case. A girl, aged seven years, admitted to the Presbyterian Hospital, October 21, 1921, had been healthy and advanced to the third grade in school. About October 1 night cries had been noted. The child would awaken out of a sound sleep and cry out several times each night. She

appeared well, however, during the day, went to school and played normally. A few days later she complained of "aching pains in the stomach" unassociated with nausea or vomiting. These recurred frequently.

October 7 a neighbor called the mother's attention to the fact that the child had a peculiar wabbling gait. She walked as if her legs were stiff. The child was kept home from school. She lay on the lounge, playing, apparently comfortable and not complaining. During the following day the symptoms increased rapidly, and October 8 she had to be assisted in walking and in attempting to stand. She complained of severe pain in the abdomen, without nausea or vomiting. Her father, who is a physician, spoke of her appearance as toxic.

A neurologic examination made by Dr. Peter Bassoe after admission to the hospital revealed a bilateral Babinski reflex, ankle clonus, weak right knee-jerk (fair on reinforcement), left knee-jerk slightly weaker and wrist and elbow reflexes present. The abdominal reflex was not elicited.

The examination pointed to a transverse lesion (partial) of the spinal cord in the upper thoracic region. The exact location of the lesion was rendered difficult because of the absence of sensory disturbances.

October 22 the mother stated that the child had had a very restless night. At intervals of from one-half to one hour, and occasionally at shorter periods, the child would suddenly awaken from a deep sleep with a cry. She stated that she did not have pain at such times. The legs became flexed, and the muscles of the lower extremities, especially those of the thigh, became firmly contracted. The spasm relaxed under massage, the patient then going to sleep with legs extended.

An examination at this time revealed tenderness over the lower cervical and upper dorsal vertebræ. On two occasions the child cried out and protested against being turned, but later allowed herself to be picked up and placed in a wheelchair. Later she turned on her left side with slight aid, and remained quiet while being examined by Dr. Bassoe.

A roentgen-ray examination, October 24, revealed a distinct but not marked change in the body of the fourth dorsal vertebra. There was a distinct area of rarefaction involving the right half of the body. The distinct outline of the body of this vertebra was broken and somewhat irregular. A diagnosis of a transverse lesion of the cord secondary to some disease process in the vertebræ was made. A positive diagnosis of the character of the bone lesion was not made.

An operation was performed, November 2. A longitudinal incision about 6 inches in length was made over the spines of the second, third, fourth and fifth thoracic vertebræ. The laminæ were removed, and a friable, vascular mass containing calcareous particles was found. This extended about 2 inches along the cord surrounding it. As much of this mass was removed as possible. The dura was not opened, for fear of transplantation of the growth within the meninges. The wound was closed in layers.

For some unaccountable reason the patient passed into profound shock. The operation was easily performed and but little blood was lost. It was thought that the patient would die during the afternoon. The temperature rose that evening to 105.6° F. The patient recovered rapidly from the shock. In forty-eight hours the temperature was normal. From November 4 on the patient made rapid progress. December 5, almost four weeks after the laminectomy, the wound was reopened, and 50 mg. of radium was placed in the region formerly occupied by the tumor. This was left in position for eighteen hours.

The patient left the hospital December 7. Evidences of return of function in the extremities with decrease of spasticity were marked at this time.

At the present time recovery is complete. The child runs, skates, goes to school each day and is practically normal. After her return home roentgen-ray treatment was continued for some time.

The tissue removed contained many calcareous particles. On section there were found numerous rather large round and

spindle cells, which formed the great part of the growth. Scattered throughout were giant cells. These were not so numerous as in many giant-cell growths. Numerous trabeculæ of bone were found. This bone was apparently newly formed, for it could not be the result of the destruction of preëxisting bone

FIG. 1.—Distinct area of rarefaction and an irregularity in the outline of the body of the fourth dorsal vertebra, indicating bone changes; the site of the lesion is indicated by the dotted line.

because of the position and different appearance of the bone that had undergone resorption. Mitoses were not seen in abundance, but there was a polymorphism about the cells and a nuclear hyperchromatism which seemed to warrant a diagnosis of giant-cell tumor. The growth was undoubtedly ossifying.

Other Cases of Vertebral Tumor. In attempting to analyze this group of cases, one encounters many difficulties. Some of the cases were reported before the knowledge of osteitis fibrosa was as complete as now, and in some cases the microscopic findings are not described as minutely as desired. I have grouped, however, the cases that have a clinical history compatible with giant-cell tumors and in which histologic examination of tissues removed at operation would seem to warrant such a diagnosis.

The number of opinions that have been expressed concerning the tissue removed from the patient on whom I operated indicates the difficulties that may be encountered in analyzing the cases. Some of the opinions will be given to indicate the difficulties of interpretation of histologic findings. It is often difficult to determine the biologic properties of cells occurring in pathologic tissues and to determine what will occur. The following are some of the opinions that have been expressed: "New bone forming in giant-cell tumor;" "benign giant-cell tumor with new bone formation;" "hemangioma;" "angioma with new bone proliferation—not atypical bone;" "chronic inflammation and repair—fairly vascular callus complicated by foreign body giant cells;" "a neoplasm suggesting benignancy composed of mesodermal elements;" "this would be called a benign giant-cell tumor, that in its new bone formation resembles a case in which the patient died of general metastases." One pathologist says: "I do not know whether it is a neoplasm or not, as I have never seen one like it; it suggests a benign giant-cell tumor with new bone in it." These opinions have been cited to indicate the difficulties that may be encountered in interpreting the character of some of these growths. The clinical course, as well as the diversity of opinion concerning the histologic interpretation, would indicate that there may be some doubt as to its nature.

Including the case that I have reported, 17 cases of primary giant-cell sarcoma or tumor of the vertebræ appear in the literature. Only those cases have been included in which a histologic diagnosis has been made. The youngest of the patients was seven years of age; the oldest, forty years.

The lesion occurred in the cervical vertebræ once, in the dorsal vertebræ six times, in the lumbar vertebræ seven times and in the sacrum once. In 3 cases no mention is made of the location of the affected vertebræ.

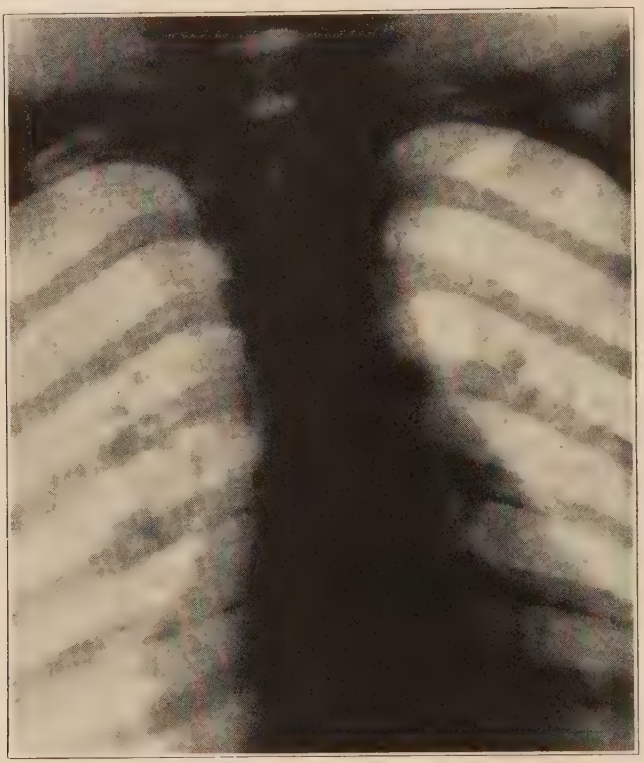

FIG. 2.—Appearance two years and nine months after the operation: Complete bone repair at the site of the former lesion, and no evidence of recurrence.

In none of the 17 cases have metastases occurred. Local recurrences have been noted. In a case observed by Madelung a recurrence was noted four years after the primary operation, and another six years later. Four of the patients died—the one just cited, operated on by Madelung, and another, operated

on by Thompson. In this case the operation was not completed because of the vascularity of the growth; the patient died some four months later. In a case observed by Leake no operation was attempted. The patient died, apparently of an ascending urinary infection. A patient observed by Cushing died four years after an operation. It is not stated whether in this case death was caused by the tumor or not.

Ossification has occurred in three of these growths. This change, occurring relatively much more frequently in these tumors than in giant-cell tumors of long bones would indicate that these lesions differ, and raises some question as to their etiology. This will be mentioned later in discussing the effects and results of intramedullary hemorrhage. In Schumann's case the growth recurred rapidly after operation, and the patient became so ill that relatives wished to take him home, thinking that he was about to die. Soon afterward the tumor ceased growing. When the patient was seen nine and one-half years later a bony mass occupied the site of the original tumor. In the case reported by Sick[1] ossification was also noted. In the history of this case it is stated that the patient was given solution of potassium arsenite (Fowler's solution).

The tumor operated on by Ashhurst recurred rapidly. Coley's toxins were then given. The tumor ceased growing and a bony mass developed at the site of the original tumor. The patient was well seven and one-half years after the treatment was first instituted.

Spontaneous cessation of growth with ossification, and ossification after the administration of an arsenic preparation, naturally raise the question whether the lesions might not have been some variety of bone cyst or a reactive tissue growth resulting from trauma. The diagnosis in these three cases in which ossification occurred was giant-cell sarcoma. It is interesting to note that an operation was subsequently performed on the patient observed by Harmer, during which tissue was removed

[1] Personal communication to Manheimer.

from the sides of the spines of the lumbar vertebræ. This was examined histologically, and a diagnosis of bone cyst made.

In three cases paraplegia has persisted, notwithstanding the fact that the growth has disappeared or ceased to enlarge. Destruction of the cord occurs unless these growths are recognized and removed, at least partially, before they invade or compress it.

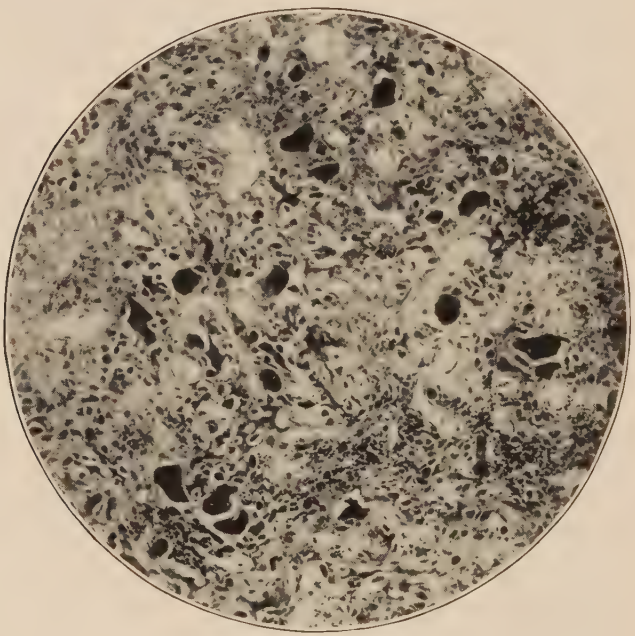

FIG. 3.—Histologic appearance of lesion.

An abstract of the histories of the cases will be given. The cases in which the patients have recovered will be considered first:

CASES REPORTED BY OTHER AUTHORS. CASE I (Madelung[1]). —A youth, aged nineteen years, had cervical vertebra involve-

[1] Riesen Zell-Sarkom der Wirbelsaule, München. med. Wchnschr., 1909, lvi, 479; a case report presented before the Medical Society of Leipzig, 1908.

ment and motor and sensory disturbances. There was a history of trauma. The patient recovered after incomplete removal with curet. The patient entered the university clinic in Strasbourg in the spring of 1905. He had been struck in the back with stone when five years old. At the present time he complained of pain in the neck when flexed, followed by motor and sensory disturbances. The patient was referred to the medical clinic at Strasbourg, where a diagnosis of tumor of the vertebra was made. Operation was performed by Madelung in May, 1905, six months after the beginning of the present illness. The tumor was found to have developed from lateral parts of the third and fourth cervical vertebræ. A thin bony shell with parchment-like crackling was exposed. When opened, a dark, bloody fluid escaped. The contents, when curetted away, formed a soft, reddish black tumor mass. A cavity the size of a small hen's egg remained. An extension toward the vertebræ, which, according to the description, was not reached, had the same contents. Histologic diagnosis made by Dr. Schmidt was giant-cell myeloid sarcoma (very vascular). There was complete disappearance of symptoms. When examined three months after operation the patient was reported as well and able to work.

Case II (Schumann).—A man, aged twenty-seven years, had involvement of the lumbar vertebræ and paraplegia. Spontaneous ossification of the tumor occurred, the paraplegia persisting. The diagnosis was giant-cell sarcoma. The patient, in May, 1898, noted pain in the back, followed by a tired feeling and sensation of weight. In June, 1898, total paraplegia, with bladder and rectal disturbances, occurred. There was some swelling over the second lumbar vertebra. A soft, rapidly growing tumor developed. In August, 1898, incomplete removal of the tumor was done; there was a rapid recurrence. In April, 1899, the tumor had reached the size of a man's head. There was marked decubitus. The patient was discharged from the hospital in a pitiable condition. He was admitted again nine and one-half years later. The growth soon ceased after the

patient's discharge from the hospital and became bony hard; decubitus healed; the bladder and rectal disturbances remained. A roentgenogram revealed the shadow cast by the bony mass.

CASE III (Manheimer[1]).—A girl, aged sixteen years, had involvement of the lumbar vertebræ (transverse processes). There was pain radiating to the lower extremities, but no cord involvement. The diagnosis was osteoid spindle-cell sarcoma with myxomatous areas containing giant cells. The patient was treated with atoxyl; she was observed only five months. Four weeks before entering the clinic she noted a swelling in the right lumbar region. The tumor enlarged without spontaneous pain. A mass the size of a fist was found to the right of the lumbar vertebræ at the level of the spine of the second Roentgen-ray examination revealed destruction of the transverse process of the second lumbar vertebra. Pain radiating to the lower extremities was noted. The tumor was removed as completely as possible, about 8 cm. of spinal dura being exposed. The patient was given, every other day, 0.01 gm. of atoxyl intravenously from May 9 to June 25. She was observed five months, at the end of which time she appeared well.

CASE IV (Sick).—There was a large fluctuating tumor between the iliac crest and the twelfth rib on the left side, and pain over the sciatic nerve and higher lumbar roots. The diagnosis was giant- and mixed-cell fibrosarcoma. A partial removal of the tumor was made. Solution of potassium arsenite was given. The patient was well four years later, a distinct shadow being apparent between the iliac crest and the twelfth rib in the roentgenogram.

There was a large fluctuating tumor between the iliac crest and the twelfth rib on the left side. The patient was considerably emaciated. Exposure of the fluctuating mass revealed a tumor which had broken through the musculature On the

[1] Ueber Heilung scheinbar inoperable Wirbelsarkome durch Arsentherapie, Beitr. z. klin. Chir., 1911, lxxii, 741.

abdominal side this had a bony capsule. Large masses of tumor could be removed. There was apparently no improvement following operative procedure. Solution of potassium arsenite was given. Believing that he would die, relatives took the patient home. Four years later the patient, when seen by Sick, was perfectly well. Ten years later the patient was alive but not well. Whether there was recurrence or not is unknown.

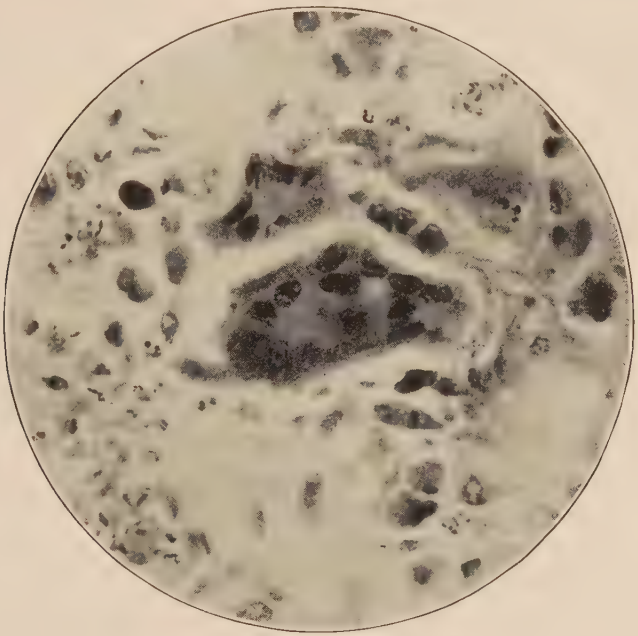

FIG. 4.—Character of giant cells and karyokinetic figures in some of the other cells in material removed at operation, as seen under high power.

CASE V (Ashhurst).—A boy, aged fourteen years, had involvement of the fourth and fifth lumbar vertebræ. After operation he was treated by Coley's toxin. Ossification of the tumor took place. Seven years later the patient was well, and served in the army. The diagnosis was giant-cell sarcoma. There was a bulging mass in the right loin of from six weeks' to two months' duration. The roentgenogram revealed involvement of the

fourth and fifth lumbar vertebræ. An incision was made, December 28, 1913; blood and tissue were removed, and the wound was packed for hemorrhage. Coley's fluid was given after three weeks, being increased gradually to 42 minims (2.6 c.c.) at a dose, every second, third and fourth day. The incision healed in a few weeks. The mass continued to increase in size, broke down in September and discharged, and then became gradually of bony-hard consistency. A roentgenogram, seventeen months after operation and seven months after cessation of treatment, revealed the shadow of a dense bony tumor. This patient was later accepted for the army and was wounded. He was perfectly well in 1920.

CASE VI (Harmer[1]).—A boy, aged sixteen years, had symptoms of involvement of the lumbar vertebræ and spinal cord. There was a history of injury three months before at the beginning of the symptoms. The diagnosis was giant-cell sarcoma. There was rapid recurrence after operation. Coley's toxins were given. Recovery followed. Later tissue was removed from the spinous process of the third lumbar vertebra, which was found to be a circumscribed bone cyst. Three months before entrance to the hospital, June 10, 1912, the patient fell, striking his spine. Pain and tenderness had persisted since. For two weeks there was numbness over the external and anterior surface of the right thigh, with loss of sensation. The patient walked with slight instability, complaining of weakness of the right knee. He stepped with great care, as the slightest jar caused excruciating pain in the lower dorsal region, shooting up to the head. A slight depression was found in the region of the tenth dorsal spine, with a swelling about the size of the hand in the subcostal region posteriorly. Both legs could be raised to about one-third the normal arc. Beyond this, pain was caused in the back. There was ankle clonus and Oppenheim's sign on the right side. There was an area of analgesia over the external aspect of the right thigh

[1] A Study of the Efficiency of Mixed Toxins (Coley) in Inoperable Sarcoma, Boston Med. and Surg. Jour., 1915, p. 61.

just below the trochanter. Roentgen-ray examination was negative. An operation performed, June 26, 1912, by Dr. Porter, revealed a bluish, rather tense but fluctuant mass beneath the muscles of the back. From this, when incised, a quantity of dirty fluid escaped. The tumor was removed as completely as possible. Bleeding was profuse. The cavity was tightly packed. The patient was put to bed in poor condition, but recuperated rapidly. Treatment with mixed toxins was begun at once, and continued for seven months; injections were given at first every other day, then twice a week. The maximum dose was 18 minims (1.1 c.c.); injections were made sixty-two times. The reaction was usually severe. There was a rapid recurrence of the tumor, $5\frac{1}{2}$ inches in length, $3\frac{1}{2}$ inches in breadth and elevated 2 inches, associated with paresis of the right leg. Injections were given in spite of the severe reaction. The tumor began sloughing in several places. In June, 1913, there was no evidence of a mass or any disturbance of sensation in the legs. Another operation was performed in June, 1914, a small mass having been discovered in roentgen-ray examination on the lateral surface of a spinous process of the third lumbar vertebra. This proved to be, when removed, a circumscribed bone cyst. There was no evidence of malignancy. The patient was well two years later.

Case VII (Rasch[1]).—The tenth and eleventh thoracic vertebræ were involved. There was compression of the cord. Pathologically, the diagnosis was a giant-cell tumor that had infiltrated the cord and spinal nerves with almost total destruction.

Case VIII (Mixter).—A man, aged thirty-one years, had involvement of the fourth and fifth thoracic vertebræ. There was no history of trauma. The symptoms were girdle pain, weakness and numbness of the legs, paralysis and sphincter disturbance. Recovery followed incomplete removal of the tumor. The patient was subsequently treated with Coley's toxins and

[1] A Case of Primary Giant-cell Sarcoma of the Spine, Hygiea, 1922, lxxxiv, 769.

the roentgen-ray. There was a complete motor paralysis and marked general sensory disturbances below the level of the fifth dorsal segment. Paralysis was more complete on the left side. The symptoms pointed to a rapidly progressing lesion at the level of the fifth dorsal segment. Complete block of the spinal canal was indicated by spinal puncture. February 14, 1922, laminectomy was performed by Dr. W. J. Mixter. The laminæ of the fourth and fifth dorsal vertebræ were removed. The bone seemed softer than normal. Overlying the dorsal aspect of the cord and to a slight degree straddling it was a rather flat, dark red vascular mass about 1 inch long. This was easily and cleanly removed from the dura by separating adhesions. A roentgenogram taken the day before the operation gave no positive indication of any abnormality. The patient was subsequently treated by the roentgen-ray and Coley's toxin.

Case IX (Coley).—A man, aged twenty-one years, before treatment was seen by Dr. Brewer, who described the tumor as looking like a soft pillow. The tumor was pronounced by Dr. Harlow Brooks to be a round-cell sarcoma with numerous giant cells. Coley classified the tumor as an atypical giant-cell tumor with local malignancy. No treatment other than Coley's toxins was given. The patient has recently been examined by Dr. Coley, and is well twenty-two years later.

Case X (Follis).—A colored man, aged forty years, had a small tumor mass near the angle of the scapula, which, in eight months, reached the size of a grapefruit, when it was operated on. The tumor recurred rapidly. A second operation was performed four months later in the patient's home. The recurrence was a large, protruding tumor, extending from the sixth to the twelfth rib. The tumor fluctuated. The skin was red and adherent. The recurrence was removed. The pathologic diagnosis was a giant-cell tumor or xanthoma. Microscopically, there were a large number of giant cells of the epulis type. The giant cells were embedded in a very cellular stroma mixed with numerous

round cells of the sarcoma type. The diagnosis, according to Bloodgood, rested between giant-cell tumor and a round-cell sarcoma containing giant cells. The patient was discharged, April 28, 1920. There was no answer to an inquiry concerning his condition.

Case XI (Cushing).—M. I., a girl, aged thirteen years, was admitted to the hospital, November 20, 1922, with the complaint of paralysis and loss of sensation in both legs. Subjective findings were complete loss of motion and sensation in both legs. Objective findings were: (1) No voluntary movement in the legs, buttocks or iliopsoas. (2) Complete loss of all forms of sensation below the level of the eleventh thoracic vertebra, with the exception of sensation in the periosteum, which was poorly localized. (3) Loss of sensation to light touch for 2 inches above the level of the eleventh thoracic as well as below; the level of loss to light touch shifted for 1 inch or 2, depending on whether stimulation came from an upward or a downward direction. (4) Loss of knee-jerks, both sides. (5) Presence of the Achilles jerk on both sides, but + on the left. (6) Flexor response to plantar reflex on both sides. (7) Protective reflex, most marked on the left. (8) Automatic bladder and rectum. An operation was performed, March 23, 1922. Laminectomy disclosed a giant-cell sarcoma, which was entirely extradural. Fairly complete extirpation of the tumor was performed. January 2, 1923, when the patient was discharged, it was noted that there was no voluntary movement in the lower extremities. The reflex of automatism was marked on both sides. Bilateral Babinski and tendon reflexes were present at both knees and ankles. There were slight clonic movements at times in the right ankle. There was no sensation except to pain on the inner side of the thigh at times. Otherwise, the patient was anesthetic below the twelfth dorsal vertebra. There was urinary incontinence. The patient voided frequently and spontaneously; this was not an overflow incontinence, though no certain method was found to cause urination. The nutrition of the lower extremities was good.

There was marked sweating of the feet and slight extensor rigidity. A healed bedsore was present. General health was excellent. The microscopic report on tissue removed was: A tumor composed of spindle-shaped cells, together with many giant cells. The nuclei of the giant cells tended to be arranged in a cluster in the center of the cell. There was extensive hemorrhage and numerous small bloodvessels throughout the tumor. The intercellular substance stained red in many areas. Small round cells were scattered throughout the tumor growth. There were several sections of normal bone tissue. The tumor no doubt arose from the vertebræ. The tumor, pronounced a giant-cell tumor, protruded between the laminæ of the tenth and eleventh thoracic vertebræ. On the left side the tumor definitely involved the laminæ. Bone and tumor were scooped out, a soft mass the size of a walnut being removed. The tumor inside the canal was grayish and surrounded the cord, extending in between the roots of the nerve. It appeared to surround the cord above the lumbar enlargement for a distance of 2 inches. It was readily removed from the dura, but with difficulty between the roots. Apparently the dura was not involved at any point. A letter, September 10, 1923, stated that sensation and motion were absent in both lower extremities, and that the patient had not regained control of the bowels and bladder.

Case XII (Cushing).—H. K. G., a boy, aged sixteen years, with a giant-cell sarcoma, was well seven years after operation. Pain in the lower dorsal and lumbar region began two years after a scuffle with another boy. The pain was dull in character, without radiation. A mass was noted in back almost one year after the beginning of severe pain. This had gradually grown larger, and was very tender to the touch. Weakness of the legs was noted. There was no disturbance of the vesical or anal sphincter. At operation all the tissues were much more vascular than normal. The spines of the eleventh and twelfth thoracic, and the second and third lumbar vertebræ were intact, though much softened. The rest of the lumbar spine was the

seat of a vascular, pulsating, soft reddish tumor covered by a thin shell of bone. The tumor passed off to the right, in correspondence with the appearance in the roentgenogram. Partial removal was made. Subsequent roentgen-ray treatments were given. The tissue was formed of cartilage and bone, with spaces in between containing a coarse fibrous tissue in which were numerous multinucleated giant cells. The bony tissue was rather intermediate between the cartilage and the bone. The tissue between the bony spicules was not very cellular and contained innumerable capillaries—osteoclasts and osteoblasts. No apparently actively growing tissue was seen.

Four patients, whose case histories follow, died. The case observed by Madelung may be somewhat doubtful, as the histologic diagnosis is not exactly clear. The tumor recurred twice during a period of ten years, but at the time of death there were no evidences of metastases.

Case XIII (Madelung).—A man, aged twenty-five years, had a tumor situated in the lumbar region, extending from the twelfth rib to the iliac crest. An operation was performed November 15, 1890. A bony shell surrounded a soft mass that resembled coffee grounds. The tumor was removed as completely as possible. A microscopic diagnosis of osteoid sarcoma with giant cells and myxomatous tissue was made. Four years later another operation was performed, and six years later still another. The patient died, but no metastases were reported; there were local recurrences.

Case XIV (Thompson).—A man, aged twenty-four years, entered the hospital with a large fungating growth over the sacrum. He was paralyzed in the lower extremities and had lost control of the bladder. No other notes are available. An operation had been previously attempted and given up because of hemorrhage. The patient died four months afterward. The histologic diagnosis in this case was giant tumor of the benign type.

Case XV (Leake).—W. H., a farmer, had been injured five years before in the region of the lumbar vertebræ. The symptoms consisted of pain and soreness in the back. The pain was thought to be due to a focal infection. The tonsils were removed with no relief. He was placed on a Bradford frame with traction. Procaine was injected in the sciatic nerve without relief. The patient later became paralyzed in the left leg, and remained so until death. Seven months prior to death he lost control of the bladder and rectum. Large bedsores developed. In the lumbar region to the left of the spine a tender spot was found, but no tumor was palpated. The patient died in April, 1923. When the necropsy was performed a hemorrhagic mass, which had destroyed the muscles, was found. This developed apparently from the first and second lumbar vertebræ, and extended 3 cm. to the left of the midline. Removal of the laminæ revealed an osteogenic tumor which arose from the transverse processes, practically filling the spinal canal. Leake was of the opinion that it was a benign giant-cell tumor which had produced paralysis, ending fatally.

Case XVI (Cushing).—R. H. C., a girl, aged thirteen years, was operated on, and a sarcoma of the sacral canal was partially removed. The principal constituent of the tissue was very large giant cells, staining feebly, and often greatly vacuolated; the giant cells were of all sizes. Death occurred four years and four months after an incomplete operation. The patient was admitted to the Peter Bent Brigham Hospital, July 14, 1915, complaining of pains in the lumbar region noted six months before admission. During the past three weeks the pains extended down the left leg to the foot, in periods. Both legs hurt. The pain was very severe when it came on. There had been rapid loss in weight. For nearly six weeks there had been some rectal disturbance. At first there was marked urgency. For three weeks there had been no sensation—incontinence, no retention. Rectal sensation was retained, and sensation was not disturbed. The patient could walk several hundred feet at a time, but had

sacral pain after sitting for a time. The tumor had perforated the anterior wall of the sacrum and filled the greatly dilated spinal canal. The patient died four years after the operation was performed. The cause of death is not given.

SUMMARY. The tumors that have been considered have some definite peculiarities. In none of these have metastatic growths been observed. Local recurrences have occurred in several, but these have been controlled rather easily by a number of different measures. In Manheimer's case the importance of atoxyl in the treatment is emphasized. In several of the cases Coley's toxins were administered. The roentgen-ray and radium have been used in other cases. Spontaneous cessation of growth occurred in the case observed by Schumann unless partial excision may be considered to have affected the growth. The tumor was, however, increasing rapidly in size when the patient left the hospital, without any evidence of cessation in growth. Recovery after partial removal or no attempt at removal has been noted in 13 of the 17 cases. In none of these has an attempt at complete removal been made, either because of the possibility of injuring the cord or because of extension of the growth into inaccessible parts.

Ossification, occurring relatively frequently in these lesions, would indicate that they differ in some respects from central giant-cell tumors occurring in long bones, although repair may occur in these following curettage; but, when this procedure is attempted, by far the greater part of the tumor is removed. Ossification occurs in bone cysts; but, judging from the description of the histologic findings in those cases in which ossification occurred, it seems certain that the lesion was a giant-cell tumor.

Because of their clinical behavior, some doubt is raised as to the neoplastic character of some of these growths, which are regarded by a few pathologists as a reactive tissue growth or an attempt at repair following trauma, in which there is formed excessive granulation tissue.

Lubarsch and Konjetzny especially have emphasized that

many of these lesions represent a reactive tissue growth following trauma. Konjetzny's conclusions are confirmed by the experimental work of Bajardi, Haasler and Enderlen. Following a hemorrhage into the marrow, the supporting tissue of the marrow proliferates to form a very cellular zone about the hemorrhage. This may form fibrous tissue to surround the hemorrhage, or invade it, transforming the coagulum into a scar. In other cases the tissue, forming as the result of a hemorrhage due to trauma, may continue to grow until tissue is formed similar to that of a giant-cell tumor. Such granulation tissue containing giant cells is found in pseudarthroses, delayed callus formation and Köhler's disease. Such reactive tissue growths may resemble histologically a giant-cell sarcoma, when biologically they should be regarded as a chronic reparative process. This excessive formation of reparative tissue occurring under certain circumstances may be compared to the keloid of the skin.

The tissue removed from the patient in the case that I reported resembles histologically that of a giant-cell tumor. The history following operation, as well as the diversity of opinion that seems to exist regarding its exact nature, suggests that the lesion may represent some chronic resorptive process with an excessive formation of granulation tissue, which is not a tumor in the strict sense of the word.

This group of cases raises some doubts as to the exact nature of many of the so-called giant-cell tumors and their biologic significance.

I am greatly indebted to those in charge of the Bone Sarcoma Registry for placing at my disposal material relating to the cases presented in this paper, and to other surgeons who have permitted me to examine material not yet published by them.

DISCUSSION

Dr. W. J. Mixter, Boston, Mass.:

Tumors of the type described by Dr. Lewis are rare and very difficult to classify.

In some 10 cases of tumor originating in the spinal column which have been in my care, there was one which was quite similar to the one described in this paper.

I feel that the whole subject of tumors arising in the vertebræ is a very confusing one, and that only by further study of this same sort will we be able to clear up the matter.

Dr. Lewis (in closing):

The case that Dr. Porter operated on and which was cured by Coley's toxin, was operated on somewhat later for what was diagnosed as a bone cyst.

END-RESULTS IN SOME CONDITIONS ASSOCIATED WITH OR POSSIBLY CAUSED BY GOITER

By MARTIN B. TINKER, M.D.
ITHACA, N. Y.

The end-results reported by C. H. Mayo, Crile, Ochsner, Frazier, Judd, Pemberton, Lahey and others have covered nearly all points thoroughly and have convinced the medical profession and the public of the value of goiter surgery, as is evidenced by the yearly increasing number of operations. There remain, however, a few questions which have been given relatively little, if any, attention in surgical literature, and to these I shall mainly devote this paper. It is perhaps unnecessary to give much time to discussion of methods of obtaining data as to end-results, yet I may mention that living in a small community, in frequent and close touch with people who are permanent residents, gives a somewhat different impression as to results than a single hurried examination or reply to a questionnaire. Our records show that of a total of 2012 cases operated upon by our present methods, approximately half come from Ithaca and a radius of one hundred miles of surrounding territory. The condition of these patients is known not only by replies to a questionnaire, but in most cases by frequent personal examination, also by reports of friends and neighbors and reports by the patients' doctors. Many in all groups discussed in this paper and all in some groups have been examined personally. The importance of reporting progress is urged upon all patients during their stay in the hospital; it is also emphasized on the printed slip giving suggestions for after-care which is handed to patients on discharge, and a geographical card index of patients helps us to get reports from neighbors who come for treatment. Most of our patients are an intelligent

class of people, who coöperate satisfactorily in reporting end-results and realize that it is frequently to their own benefit, as well as others similarly afflicted.

Pregnancy. A number of times every year I am asked: "What would be the probable effect of pregnancy on the results of operation for goiter; would thyroid enlargement and the symptoms probably return as the result of pregnancy? Would the child show any physical or mental abnormalities as a result of thyroid lack in the mother? Would iodine, thyroid extract or other medication be desirable during pregnancy?" The impression seems quite general that pregnancy generally does precipitate hyperthyroidism or induce permanent thyroid enlargement, or both, but very little has been reported from the basis of actual clinical experience on this subject. I have reliable information as to the condition of 12 patients, pregnant at the time of or subsequent to partial thyroidectomy; all have gone through their pregnancies to full term and all have given birth to healthy children. One has given birth to 3 children, 2 have given birth to 2 children; 8 have given birth to 1 child since operation: a total of 15 children. These patients were operated upon from three to eleven years ago, and only 1 has required further surgery. This patient was first operated upon at the time when most of us were doing hemithyroidectomy because of fear of postoperative cachexia strumipriva. She had considerable enlargement of the left side at the time of the first operation, and returned because of further enlargement and pressure symptoms. A further partial thyroidectomy was done from which she has made a satisfactory permanent recovery, now four years past her final operation. Another patient, personally examined a few days before writing this paper, has a slight enlargement which does not warrant operation at this time. The remaining 9 remained apparently entirely well. Five of the number originally had disfiguring or obstructive growths; 3 were toxic adenomatous goiters; 1 very toxic exophthalmic. Four were examined as to end-results personally, 1 by an assistant and 4 reported by letters. Of 3 patients who were pregnant at the time of

operation, 1 was operated upon at three months because of obstructive symptoms; another was operated upon at seven months because of suppurative thyroiditis; attempts at relief by drainage without radical surgery were unavailing and most of an extensively infiltrated gland was removed with considerable difficulty; recovery was rapid in both cases and they remain well three and five years after operation. The third patient had a highly toxic exophthalmic goiter and elected partial thyroidectomy rather than abortion at five months; her pregnancy went to term and she gave birth to a healthy child, and has since safely gone through another pregnancy; both of her children are normal and she remains in good health eleven years after operation in spite of the somewhat trying conditions of the life of a missionary in China.

So far as can be judged from so small a number of cases, the end-results seem to justify the conclusion that thyroid surgery need have no evident effect on children born at a subsequent pregnancy; that there is not much tendency to the return of symptoms or enlargement; and that operation during pregnancy is not particularly dangerous.

Menstruation. As to menstrual function: There is complete cessation of periods for from three to six months or longer in practically all of the *extremely* toxic adenomatous and exophthalmic goiters which we see. In no instance under my observation has this been permanent and the return of menstruation occurs as soon as the patient is well on the road to recovery. At first most patients are greatly disturbed over the cessation of menstruation, and inquiry as to the probable outcome must come to almost everyone who sees many toxic goiters.

Profuse menstruation as the result of lack of normal thyroid secretion has not been reported to us in a single instance. The danger of myxedematous changes as a result of thyroidectomy is probably greatly overestimated. Osler, who no doubt had under observation as large a number of patients as any consultant of his time in this country, in at least two editions of his text-book, stated that postoperative myxedema is very infre-

quent, that he had observed only 2 such cases, and that 1 of these cleared up under treatment. Probably surgical methods and results have greatly improved since this statement was made.

Exophthalmos. My impressions as to ultimate results with exophthalmos have radically changed within a few years. The results as regards appearance of the eyes following operation upon patients with extreme exophthalmos are often discouraging even after four or five years have elapsed. As with other symptoms, the cases of long standing improve more slowly and less surely, but observation of late end-results leads me to believe that in many cases exophthalmos continues to lessen, even long after the general physical condition has come back to normal. In 39 cases examined by me or my assistants seven to fourteen years after operation, the eyes of 31 are normal, or so nearly so that they would not be noticeable to the average observer. All of these patients live near Ithaca and most of them have been seen frequently. Few of these extreme cases showed much improvement in the first three years and some retained noticeable exophthalmos for five years. While I formerly told patients with very prominent eyes that the prospect of improvement was doubtful, on the basis of observation of later end-results, I now tell them that they have at least three chances out of four of ultimately getting fairly normal eyes.

Advanced Age. What should be advised in the case of an aged person suffering seriously from goiter is often a difficult question. The distress caused in some of these cases is indicated by the fact that patients as old as eighty-six have come for consultation to consider surgical relief. Of course, no sane surgeon advises operation for any condition with a patient advanced in years unless the indications seem urgent, hence, experience as to end-results in older goiter patients is limited. The widespread impression, both in and out of the profession, that lack of thyroid secretion influences the development of senile changes has doubtless led most of us to be especially slow to meddle with the thyroids of older people. Yet my own experience in operating upon 31 patients over sixty years of age, who we have been able

to follow, leads me to feel that possibly there is quite another side to the question. The indications for operation seemed definite in all of these cases: 19 had obstructive symptoms, 11 had adenomata with quite a high degree of toxemia, and 1 was malignant. Of 15 patients between sixty and sixty-five years of age, 3 have died: 1 from local recurrence of malignancy (small round-cell sarcoma); 1 from operation at another clinic for enlarged prostate; and 1 from apoplexy. The remaining 12 are living and well; 8 of these patients are from five to eleven years past operation; 1 patient, now seventy-one years of age, is nine years past operation; 1, seventy-six years of age is eleven years past operation. Three patients were over sixty-five years of age at the time of operation; 1 of these patients died from unknown cause; 1 is living at sixty-nine years of age; 1 patient operated upon for serious obstructive symptoms when seventy-two years of age was living at last report, with failing eyesight, but otherwise well, over eighty years of age. Many of these patients looked thin, almost to emaciation; pale, gray and even older than their years. Many of them looked ten years younger before leaving the hospital and all considered themselves greatly benefited. Is it not possible that the drive of an overactive thyroid for years causes rapid wearing out quite as frequently as thyroid lack precipitates senile changes?

Childhood and Adolescence. The important influence of the thyroid in development, both physical and mental, rules out surgery in childhood and adolescence except in cases presenting urgent symptoms, but harmful results have not appeared in any of my patients operated upon when under twenty years of age. Twenty-eight cases are included in this group. Avoidance of unfortunate end-results seem to me to be dependent upon leaving a fairly liberal strip of thyroid tissue with unimpaired blood supply from at least two of the four main arteries. Dr. W. S. Halsted's early work showed conclusively that dogs with a small amount of thyroid, with normal blood supply at one pole, are capable of hypertrophying the stump to supply the needs of the animal.

The strip saved should be along the posterior surface of the gland over the great vessel sheath and along the trachea and larynx in order to avoid injuring the parathyroids and the recurrent nerves. With this precaution none of my younger patients have developed any evidence of thyroid or parathyroid lack. The youngest patient was a little girl, operated upon at three and a half years of age, because of serious symptoms of obstruction in breathing. She made a perfectly satisfactory recovery, and at last report was well eight years following operation. A girl, aged fourteen years, operated upon because of an extremely toxic exophthalmic goiter, has remained entirely well fourteen years since operation, the longest of any of my children patients. She had developed from a child into a very attractive young woman, has taken an art course with credit and is now a successful illustrator, is married and remains entirely well.

Glycosuria. Approximately 5 per cent of all toxic goiter patients coming under my care have a trace of sugar in the urine. In all cases these small traces have cleared up with attention to diet, and recently occasional use of insulin, while the patients have been in the hospital, and the end-results three to fifteen years after operation show no evidence of trouble from this source. There are 53 patients in this group. On the other hand patients with high percentages of sugar in the urine and high blood sugar associated with toxic goiter usually do badly. Three such patients have died without any surgery within a week of admission to the hospital, in spite of the best medical care available. Three have been sent back to their home physician. Another three, exactly $33\frac{1}{3}$ per cent in my limited experience, have been brought sugar-free under medical care, have had preliminary ligations followed by later excision and remain well without very strict medical supervision, although they are requested to report regularly for urinalysis and occasionally blood-sugar estimation. To summarize my own experience: The lower percentages of sugar have cleared up before operation, remaining permanently free after operation, without special attention to diet or medication; in case of high percentages of

sugar associated with toxic goiter two-thirds of the patients die without operation; while when higher percentages are cleared up by diet, insulin or a combination of the two sufficiently to warrant operation the patients do well, requiring little medical attention to keep them sugar-free.

High Blood-pressure. Several writers on blood-pressure state that the administration of thyroid extract usually causes a fall in blood-pressure, although in certain instances blood-pressure is increased. This belief that thyroid secretion usually lowers blood-pressure has probably led to the assumption that removal of goiter would have an unfavorable influence on high blood-pressure, for, of course, a certain amount of normal thyroid tissue is removed, even with large adenomatous and colloid goiters. As a matter of experience, dangerously high blood-pressures are frequently favorably influenced by thyroidectomy. Until recently I have not operated upon patients with very high blood-pressures, hence, the end-results do not date back in most of my cases more than to five years. Patients with blood-pressures over 180 are invariably given preliminary treatment—rest in bed, low protein, salt-free diet and free purgation. In the case of patients with pressure over 200 systolic, we have not operated unless it was possible under treatment to reduce the pressure to 190 or lower. In 11 cases with pressure above 200 systolic, there has been such immediate and striking improvement after thyroidectomy that it seems fair to attribute the patient's improvement to operation. In one instance the blood-pressure dropped immediately from 200 to 150, and later was reported by the patient's home physician at 120; in another instance the drop was from 220 to 140; a third patient dropped almost immediately, following thyroidectomy from 240 to 150, and still further improvement has been reported since this patient returned to her home. The less encouraging side of the question is that 2 patients have died from apoplexy within a few months after leaving the hospital, and in the remaining 20 patients the improvement has not been greater than would be expected from such restriction of diet and general care as it

has been possible to enforce. Thus far we have been unable to discovery any criterion enabling us to select those patients with high blood-pressure who would be benefited from those in whom no improvement could be expected. There has been unquestionably a very striking immediate and thus far permanent improvement in 30 per cent of the high blood-pressure cases. If a three years' period may be considered an end-result the end-results are good in this proportion of my cases. It seems to me that this condition is deserving of careful study by laboratory as well as clinical workers.

These are but a few of many unsettled problems which must come to any one seeing a considerable number of goiter patients. The wide experience of many members of this Association should add much toward their solution.

END-RESULTS OF GOITER OPERATIONS

By A. J. OCHSNER, M.D.
CHICAGO, ILL.

The following plan was carried out in order to determine, as nearly as possible, a fair idea of end-results in this type of cases:

A questionnaire, containing the following inquiries, was sent to the address of 1200 patients upon whom Dr. N. M. Percy and I had operated at the Augustana Hospital for exophthalmic and toxic goiters during ten years, ending January 1, 1923, so that none of the answers included came from patients operated less than fifteen months ago:

1. Have you been well since returning home from the hospital?
2. How long were you weak after returning home from the hospital?
3. Have you followed directions on the diet list?
4. Have you continued drinking boiled or distilled water?
5. Have you any disturbance of the heart?
6. Have you gained in weight?
7. Have you had any new complaints since leaving the hospital?
8. Please give a general description on space below of how your health has been since leaving the hospital?

The following table gives an analysis made by our colleague, Dr. O. E. Nadeau, of the answers received from the first 500 patients who sent in their reports. Later the remaining answers also will be analyzed. It seemed sufficient for the present paper to analyze this number:

ANALYSIS OF 500 REPLIES TO QUESTIONNAIRES IN CASES OF TOXIC AND EXOPHTHALMIC GOITER

Ages.	Cases.
11 to 20 years	48 (9.6 per cent)
21 to 30 "	129 (25.8 ")
31 to 40 "	123 (24.6 ")
41 to 50 "	105 (21.0 ")
51 to 60 "	73 (14.6 ")
61 to 70 "	19 (3.8 ")
71 to 80 "	3 (0.6 ")

DURATION OF WEAKNESS AFTER OPERATION

1 month	154	1 year	42
2 months	96	2 years	24
3 "	69	3 "	3
4 "	27	4 "	3
5 "	12	5 "	1
6 "	36	6 "	2
7 "	7	7 "	0
8 "	3	8 "	1
9 "	2	9 "	1
No mention	17		

Symptoms recurring after operation similar to some of those experienced before operation, 119.

Those who followed directions, 278, but symptoms recurred in 73.
Those who did not follow directions, 207, but symptoms recurred in 41.
Those who drank boiled water, 154, and symptoms recurred in 38.
Those who did not boil water, 337, and symptoms recurred in 77.
Those who had recurrent symptoms, but did not mention diet, 5.
Those who had recurrent symptoms, but did not mention water, 4.

Disturbances of heart: Yes, 125; no, 357; no mention, 18.

Voice affected temporarily, 5; permanently, 1.
Recurrent tumor of neck, 35.
Persistent exophthalmos, 44.
Worse after operation, 8.

DEATHS

2 months, 1.
7 months, 1.
9 months, 1.
2 years, 2 (of these 1 died of carcinoma of the stomach).
3 years, 3 (of these 1 died of suicide from melancholia).
4 years, 2 (of these 1 died of diabetes).
5 years, 1.
6 years, 2 (died of diabetes).
9 years, 1.
Patients who stated they felt better when following strict directions, 21.

UNUSUAL COMPLICATIONS

1 case has unilateral exophthalmos (dextra) six years after operation.
2 cases (male) developed insanity one and a half years after operation.

It will be seen that 70 per cent of our cases were between the ages of twenty and fifty years and that 64 per cent had regained approximately normal strength within three months following the operation, while 15 per cent remained weak for more than one year.

In 24 per cent some of the symptoms present before the operation had either persisted or they had recurred since the operation, but with the exception of the cases noted especially below, almost all of this group of patients were better in some respects than they had been before the operation, although none of them were entirely well.

During this period of ten years we have given each patient the following printed list of directions upon leaving the hospital, with the hope of improving the prognosis:

1. Avoid excitement or irritation of every kind. If anything happens to annoy you, put it off for a week. Never do anything in a hurry or long enough to become really tired.

2. You should get an abundance of rest by going to bed early, not later than 9 P.M., and taking a nap after luncheon.

3. You should get an abundance of fresh air, especially at night, consequently you should sleep with wide-open windows or on a sleeping porch.

4. You should drink nothing that irritates the nervous system, like tea, coffee or alcohol. Of course, you should not use tobacco in any form.

5. You should eat very little meat. If you are very fond of meat, take a little beef, mutton or breast of chicken or fresh fish once or twice a week, or at most three times a week.

6. You should drink a great deal of milk or eat things that are prepared with milk, such as milk soup, milk toast, etc. Cream and buttermilk and Horlick's malted milk are especially good for you.

7. You should avoid beef soup or beef tea or any kind of meat broths.

8. You should eat an abundance of cooked fruits and cooked vegetables of every kind, or very ripe, raw fruits, or drink fruit juices prepared out of ripe fruits.

9. You may eat eggs, bread, butter, toast, rice and cereals.

10. You should drink an abundance of water boiled for twenty minutes (distilled water may be used in place of boiled water). Do not drink unboiled water.

The table shows that the majority of these patients have not followed these directions constantly since leaving the hospital, and that there was practically no difference in the percentage of complete cures between those who did and those who did not constantly follow these directions, except that 21 patients stated definitely that they felt better when they followed the directions strictly.

Twenty-five per cent of our patients still suffer to some extent from tachycardia or some other form of cardiac disturbance.

Five patients (1 per cent) had some disturbance of the voice for a time, which has, however, completely disappeared, while in 1 patient it has persisted to some extent.

There was a recurrence of the goiter in 7 per cent of the cases, and all of these had returned to their former mode of living, including the drinking of unboiled water.

Exophthalmos persisted in 9 per cent of the cases. In 1 case a right-sided unilateral exophthalmos has persisted for six years. Three cases died within one year after the operation. Twelve died from two to nine years after the operation from diseases not connected with their goiters. Aside from these only 8 cases (1.6 per cent) of all the cases were worse after the operation than before.

It has been possible to make a personal examination of only a small proportion of these patients in order to compare their present condition with the records of their preoperative condition, which would, of course, be necessary to make a report of end-results satisfactory.

All of the cases that I had an opportunity to examine personally showed a marked improvement over their preoperative condition, even in case of the presence of some unfavorable symptoms, but this may have been a simple coincidence and may not hold true for those not personally examined.

REPORT OF THE RESULTS OF OPERATION ON A GROUP OF 150 CASES OF GOITER

By CHARLES N. DOWD, M.D.
NEW YORK

The effort of the Executive Committee of the American Surgical Association to promote late reports on the results of treatment for goiter, is surely a commendable one. There is hardly a subject which is more written about in surgical literature than goiter, and the effort to solve the goiter problem is attracting the energies of a very large number of competent men; but in proportion to this great activity, there has been too little attention to the late results of the different forms of treatment.

Wishing to add a little to the subject, I have endeavored to study the later condition of 150 patients, on whom I have personally operated for goiter between 1899 and 1924. The report is confined to personal cases, because they give better opportunities for study than we are able to obtain from groups which represent the work of several surgeons on a hospital division. They represent all the patients on whom the writer has operated for goiter during this period.

In addition to these cases many others have been treated by medication or rest or hygienic *regime* or by *x*-ray. Most of them have passed out of observation, but have shown certain peculiarities which are referred to in the text.

The types of goiter have varied from acute hyperplastic goiters of overwhelming toxicity to encapsulated cysts with little or no toxicity. Toxic adenomata, however, were more common than any other type.

There were also 2 cases of cancer which are not included in the list, because only diagnostic incisions were made.

All but 5 of the patients have been traced since leaving the hospital. The periods of observation are indicated in the following table:

Followed into 21st yr.	1 patient	Into 9th yr.	9 patients
Followed into 20th yr.	1 patient	Into 8th yr.	13 patients
Followed into 17th yr.	1 patient	Into 7th yr.	8 patients
Followed into 16th yr.	2 patients	Into 6th yr.	14 patients
Followed into 15th yr.	4 patients	Into 5th yr.	17 patients
Followed into 13th yr.	2 patients	Into 4th yr.	14 patients
Followed into 12th yr.	4 patients	Into 3d yr.	16 patients
Followed into 11th yr.	3 patients	Into 2d yr.	16 patients
Followed into 10th yr.	7 patients	Into 1st yr.	8 patients

The reports have been made by the writer from personal observation in 83 instances; by another physician 11 times; by the patient either by letter or telephone conversation with the writer 26 times; by a nurse 6 times; by the patient's relative or friend 14 times.

Kinds of Operations. The operations have been done primarily with the view to adjusting the operative procedure to the patient's strength, but with the ultimate purpose of removing as much of the thyroid gland as the patient could spare without endangering the normal thyroid function. In most instances, four-fifths of one lobe and from one-third to three-fourths of the other lobe have been removed. A primary ligation of one or both superior thyroid arteries has been done in 12 instances. Usually this has been followed by the removal of a large portion of the enlarged gland. Occasionally no further operation than the ligation has been practicable or even desirable. As the work has progressed, it has been possible to gauge the patient's strength in such a way as to keep within fairly safe limits in the selection of operative procedure.

Standards for Estimating Results. In estimating the results of these operations, we have based the reports on the ability of the patient to perform the ordinary duties of life. We believe that this is the most important standard. The patients who come to us with goiter wish to be cured of their disabilities, so as to

have at least the ordinary capacity for work and enjoyment. A result short of this is not satisfactory, no matter what the pulse-rate or basal metabolism or bodily weight. If, however, these patients are able to carry on the ordinary duties and pleasures of life in comfort and health, the results may be considered satisfactory.

It is important to have a mental picture of the entire group of patients, thus endeavoring to show what may be expected of such people after their operations.

We have, therefore, classified them into five groups:

1. Operative fatalities.

2. Patients who have died either of intercurrent disease or from results of goiter since leaving the hospital.

3. Patients who are definite invalids.

4. Patients who are able to do a moderate amount of work and to enjoy life, but habitually have to guard against overexertion.

5. Patients who really enjoy life and have at least the average capacity for work or other activities.

Fortunately, the latter group preponderates in a large majority. If we take the entire group of patients who have been observed since leaving the hospital, 73.6 per cent belong to this group. An additional 22.8 per cent are grouped with those who can enjoy life and do a moderate amount of work, but have to guard against overexertion.

In order to explain these divisions, we may refer to some of the patients in detail.

Division 1. *Operative Fatalities.* Five patients. The first 3 had advanced Graves' disease in its extreme form. Their ages were, respectively, twenty-three, twenty-six and sixteen years. They had exophthalmus, palpitation, sweating, extreme nervousness and very rapid hearts. One of them died on the table before even a ligation could be completed. Another died three days after operation with edema of the lungs and the other on the third day after operation, having "rusty" sputum and signs of pneumonia.

In view of subsequent experience, operation would not have

been attempted on these patients in the conditions which they then showed. These 3 fatalities occurred among the first 33 cases. It is possible that a preliminary ligation of a single superior thyroid artery at a properly selected time without even removing the patient from bed might have been done successfully for one or all of them and that further procedure could have been accomplished later.

It is to be noted that in the next 117 cases there were only 2 deaths. One of them, a girl, aged twenty-three years, who had suffered from hyperplastic goiter for several years and had undergone several operations, died after an effort to remove a part of the remaining left lobe of the gland. The other was a patient who besides her goiter had mitral regurgitation and a dilated heart. She was kept in the hospital for twenty days before operation, and although her heart lesion was well understood, the operation seemed advisable. She went through the operative procedure without incident. Her pulse was about 90 throughout the operation, and she seemed to do well until the evening of that day, when she suddenly died. The physician who observed her at that time thought that her death was due to a coronary embolus. Every surgeon who is not excessively timid has to take an occasional risk of this sort. When, however, we consider that there were 117 consecutive cases with only these 2 deaths, we may appreciate that we are dealing with a very low operative mortality.

In considering this group of fatalities we are impressed with the deadly character of the disease in the first 4 of these patients and our great regret is that their thyroids could not have been attacked before their hyperplasia had led to such serious symptoms.

Division 2. *Patients Who have Died Since Leaving the Hospital, Either from Progress of the Goiter or from Intercurrent Disease.* Four patients: One, aged thirty-three years, who had suffered from goiter for seven years and who had reached the stage of degeneration of her internal organs. She was kept in the hospital for twenty-nine days, resting in bed and having symptomatic medical treatment. Her basal metabolism varied between 64

and 39. The superior thyroids were then ligated without incident, one at a time, under local anesthesia, in her bed. She improved considerably, but insisted on going home. She died a month later in another hospital from so-called "heart disease."

The second case was a girl, aged nineteen years, with a moderate-sized goiter, exophthalmus, basal metabolism plus 37 and hereditary syphilis. Her two superior thyroids were ligated, and she improved satisfactorily, so that at one time I was willing to undertake subtotal thyroidectomy. Her guardians, however, refused this and she went from the hospital. She was under *x*-ray treatment for more than a year. She finally died with apparent degeneration of her internal organs.

The third case died, about a year after leaving the hospital, from "cerebrospinal meningitis." Her physician stated that she did not then give signs of hyperthyroidism.

The fourth case had both superior thyroids ligated in March, 1917. No further operation was done at that time. She was then extremely toxic. She existed for several years in a condition of invalidism, going from one hospital to another, having long treatments of *x*-ray, etc. In February, 1924, an effort at operation was made in another hospital, and she died immediately following the operation.

It should be noted that ligation of the superior thyroid arteries was successfully accomplished for 3 of these patients, and that they then either would not or could not have further surgical procedure, and that the disease progressed in each instance. The fourth apparently died of disease not associated with her goiter.

DIVISION 3. *Patients of the Invalid Class.* I find only one patient in this group. He, too, refused further operation after his superior thyroid arteries had been successfully ligated. He is a man, aged twenty-six years, who was first seen six years ago, having then had his goiter for three years. He had exophthalmus, tremor, weakness, extreme "nervousness," sweating and rapid pulse. Both superior thyroid arteries were ligated. Improvement followed, but he refused further operation. He tried to

work as a clerk, but was unable to continue. In February, 1920, the right inferior thyroid artery was ligated by another surgeon. He then made another unsuccessful effort at light work. He was in another hospital for six weeks in 1921. He was seen in February, 1922, and then showed weakness, sweating, dilated heart, palpitation and tremor; his neck was $16\frac{1}{2}$ inches in circumference. He was then three months in another hospital and had twelve *x*-ray treatments; improvement followed. He then tried again to do clerical work, but when seen by his physician, March, 1924, had been unable to continue even light work. It is hardly fair to consider him a case of operation. He let his opportunity pass when he refused partial thyroidectomy after his preliminary ligations.

Division 3. *Patients Who Are Able to do a Moderate Amount of Work and Enjoy Life, but have to Guard Against Overexertion.* There were 32 patients in this group. They were all definitely better than before their operations. Several of them were better than we could fairly expect. Three cases may be cited for illustration, although others in the group were much stronger than these were. A woman, aged thirty years, showed marked invalidism from the ordinary symptoms of toxic goiter. After subtotal thyroidectomy and a period of rest in the hospital, she regained her health and seemed to be normal for nearly four years. She then suffered from a postpartem hemorrhage. During the following two years her strength has only been moderate, but she has been able to carry on the ordinary duties of life. Another patient, aged twenty-eight years, with severe Graves' disease, had her superior thyroid arteries ligated separately under local anesthesia in 1912, with an interval of seven days between the two ligations. This was followed four months later by hemithyroidectomy. During the following six years she was not able to do much. During this period her disability was increased by family sorrow. She then gained strength, and during the intervening years has been able to support herself and help care for her mother.

Another patient, aged forty years, made an excellent recovery and was able to return to her usual family and social activities.

There followed a period of stress owing to misfortune in the family and corresponding period of depression and limitation of capabilities. Now for three years she has been reasonably active and care free.

These cases represent the least capable part of the group. Without the operation, I believe that most of them would have collapsed under their trials or would have been useless invalids.

Division 5. *The Group of Patients Who Really Enjoy Life and have at Least the Average Capacity for Work or Other Activities.* This is by far the largest group, representing, as before stated, 73 per cent of the patients who were traced after leaving the hospital, or 68 per cent of the entire series, including untraced and fatal cases. Many of them have much more than the average capacity for exertion. For instance, Case 94 announced that "before operation" she could not climb stairs or raise her arm high without distress or "do much of anything," but that now, at the age of forty-nine years, five years after operation, she can do more than she had ever been able to do before, attending to her own housework and taking part in various activities. Case 59 reported eight years after her operation that she is caring for four children and husband, a big house and garden, and stated with much pride that on the day before she had washed twenty-two windows. Her goiter was large and contained an unusual combination of soft cellular and firm, fibrous material.

Case 58 has carried on a life of unusual intellectual activity as a writer during the eight years which have elasped since her operation, although she has suffered from a broken hip with its necessary confinement during that period. Her operation was for cystic adenoma of fifteen years' duration and two years' rapid growth. At the time of her operation her pulse was 120, and she suffered from palpitation, tremor and perspiration.

Case 12, fifteen years after an operation, "tends store all day, beginning at 6.30 A.M., and does the housework for herself and mother before and after these duties." Her operation was for a very large adenoma of fifteen years' duration and two years' rapid growth, severe pressure symptoms and moderate constitutional disturbance.

These facts are given in detail in an effort to show to others the impression which we have received on following up these patients. This group has impressed the writer as showing more than the average degree of good health and capability. All of them were incapacitated before operation—most of them from "toxic" symptoms—a few of them from pressure symptoms. Their study indicates a remarkable average of good health after previous disabilities which had been disturbing in a high degree.

It should be stated that in no instance has operation been done for the simple goiter of adolescence or for any other form of simple goiter which did not give definitely disturbing symptoms.

Of late much attention has been given to the query as to how patients with goiter will do without operation or even without treatment.

The histories of some of the patients mentioned in Groups 1, 2 and 3 indicate the condition to which such patients may drift. Two other experiences which throw light on the subject also come to mind: One, a woman, aged fifty-eight years, who had a goiter for thirty years. Her pulse had frequently been rapid and she had frequently been obliged to stop her labors at housework, but she would always resume them again as soon as the acute attack had passed away. When I saw her she had acute pain in the epigastrium. Her heart was dilated, very irregular and gave beats from 160 to 180 per minute. It was of extremely poor quality and made any operative procedure out of the question. She was very thin and had a large goiter, most marked on the right side of her neck and apparently cystic there. This was an example of chronic goiter which had finally resulted in cardiac failure. She died on the following day. The other, a man, aged forty years, who had lived in the Middle West, and had been suffering from the symptoms of acute thyroid disease for three months. He had lost 20 pounds, was weak and had to give up work. He had a severe tremor and rapid pulse and was very nervous. His goiter was soft and of moderate size. He was taken to the hospital for a more thorough examination, but became restless after two days and returned home. His weakness

then increased and he died within a few days. All surgeons who see many goiters have similar experiences and are forcibly impressed with the seriousness of the disease in both its chronic and acute forms.

When such cases are contrasted with the results which have followed the operations in this group, one must feel like attacking the disease in a radical method.

Cancer or Suspected Cancer. The pathology of the thyroid is complex. It brings puzzling problems to both pathologists and clinicians. The most remarkable instance in this group occurred in a man, aged forty-eight years, who for a year had suffered from a left-sided goiter, with both constitutional and pressure symptoms. An encapsulated cystic tumor, 9 x 7 x 5 cm. in its diameters, was enucleated from the left lobe of the thyroid. None of the adjoining tissue was removed. Since there was no apparent extension of the growth beyond its capsule, and since its enucleation was easy, malignancy, was not clinically thought of. However, three well-known pathologists agree in the diagnosis of "adenocarcinoma, probably of low malignancy." At the present time, three and one-fourth years after the operation, the patient is free from recurrence and is enjoying excellent health. Another patient who had a papillary cyst adenoma has now been followed for nine years. He is entirely free from symptoms and is actively engaged in business. He had rapid recurrences after his first partial operations, and finally every discoverable bit of thyroid gland was removed and radium was left in the wound for a few hours, in the endeavor to control any fragment which might possibly remain. There was also much pathological and clinical consultation about this case, and bad prognoses were generally given. There were two other papillary cyst adenomas in the group, but there was no recurrence or suggestion of malignancy in either of them. Another tumor, which clinically suggested a progressing neoplasm, was reported "lymphogranuloma of the thyroid." The patient made an excellent recovery after its removal, and now, in her fifth postoperative year, shows no suggestion of recurrence. These cases are mentioned to show the uncertainties of thyroid pathology.

The relationship of the x-ray to the thyroid gland should also be considered. A number of cases here recorded had received *x*-ray treatment before coming for surgery. It is not now possible to say how many. Some of the cases mentioned in Divisions 2 and 3 also received *x*-ray treatment after leaving the hospital. I have had at least four patients treated by *x*-ray without operation. Two of them were very mild cases and have done well. A third case was toxic; when first treated he had a marked tremor, basal metabolism plus 35, with a very small thyroid. He did well under *x*-ray treatment. Another one, with a large adenoma and very mild toxic symptoms, preferred *x*-ray treatment to operation, and now, after the lapse of a year, is in about the same condition as when first seen.

It is notable that experienced roentgenologists consider hyperplastic thyroids the most suitable type for *x*-ray treatment and that many failures of relief come after *x*-ray treatment of such goiters; also, it is notable that adenomata which are not so well suited for *x*-ray treatment produce some of the most disturbing symptoms and respond particularly well to surgery.

The results of ligation of the thyroid arteries have been interesting. Preliminary ligation of the superior poles of the thyroid gland, including the arteries, has been a common procedure in patients too ill for more extensive primary operation. Definite improvement almost always follows this procedure, so that after two or three months a suitable portion of the gland may be removed. These patients, however, are not always easily controlled and sometimes cannot be persuaded to submit to the secondary operation. Cases already quoted in Groups 1 and 2 show the unfortunate condition into which such patients may drift. One patient, however, did so well after double superior pole ligation that secondary operation was not advised, and now, after the lapse of eleven years, is in excellent condition. She was a young woman, aged twenty-four years, with a short history of acute hyperthyroidism.

There are two notable results following quadruple ligation: One, aged thirty-eight years, had severe toxic symptom of five months'

duration. She had a rather large symmetrical goiter, exophthalmus, tremor, pulse 120 to 150, and was too weak to work. The superior arteries were ligated in June, 1919, and the inferior arteries in February, 1920—she made a remarkable recovery and has now been working successfully as laundress for more than three years. Another young woman whose symptoms were complex, but who had exophthalmus—moderate-sized goiter, rapid pulse, "nervousness," inability to work and basal metabolism + 17, recovered her poise after quadruple ligation and at the present time, three years after her first ligation, is successfully conducting a boarding-house.

Operation on patients under the age of twenty-one is not often desirable. The goiter of adolescence frequently subsides without operation. There are two rare exceptions to this rule: First, the young patient who develops extremely toxic symptoms—"true exophthalmic goiter;" second, the young person who develops an adenoma so large as to give pressure symptoms or real disfigurement. An instance of the latter condition may be referred to. Case 16 came to me in 1909, when twenty years of age, having a goiter which had been present twelve years. It was very large and was giving great disfigurement and some pressure symptoms. I removed a large part of it and found it to be an adenoma with cystic formation in some parts. On looking her up, fourteen years later, I find her in excellent health. She has had seven children in the meantime and her goiter has returned. Her neck is now the site of a large cystic goiter, which is similar to the one which was removed fourteen years ago. However, it is soft and does not give pressure symptoms.

There have been a few other adenomas in young girls which have developed to considerable size and have given definite pressure symptoms. These have been removed, with good results.

The seriousness of long persisting adenomata and the favorable result of operation may be illustrated by the following case: A patient, aged forty-five years, with a moderate-sized goiter, came to the hospital with a six-year history of diability, having had remissions and exacerbations, but having endeavored to carry on her ordinary duties during most of the period. She

has recently shown lack of strength, tachycardia, tremor, nervousness and perspiration, and was unable to work. On admission to the hospital her disability was extreme. Perspiration and weakness and nervousness were excessive. Basal metabolism was +83. She manifestly was not in condition for surgical procedure. She went to an endowed room in the medical division for five weeks, where Dr. Sumner kindly attended to her treatment. She showed considerable improvement, her basal metabolism went down to +30 and then went to +40. Her heart was dilated and rapid and irregular, pulse being 110 to 120. She gave definite evidence of degeneration of the cardiac muscle. However, since the thyroid nodule was apparently encapsulated, and since she had ceased to improve under medical treatment, I removed the nodule by a very short operation and she made a remarkably satisfactory recovery, pulse coming down to 64, basal metabolism reaching normal and her entire mental attitude being satisfactory. Her strength improved and she was able to return to her ordinary duties.

Recurrences. In 1 instance (just referred to) there has been a recurrence of an adenoma which has reached a large size. In 7 other instances there have been moderate recurrences of adenomata, none of them serious.

Hypothyroidism has not been observed in any of the patients.

SUMMARY. 1. The results of treatment on these 150 patients may be tabulated as follows:

	Number of patients.	Percentage of those patients who were traced after leaving hospital.	Percentage of entire group.
(*a*) Enjoying good health and able to do at least the ordinary amount of work	103	73.6	68.66
(*b*) Able to enjoy life and do moderate work, but carefully avoiding over-exertion	32	22.8	21.33
(*c*) Persistent invalidism	1	0.7	0.66
(*d*) Died since leaving hospital	4	2.9	2.66
(*e*) Died in hospital	5	....	3.33
(*f*) Not traced	5	....	3.33
Total	150		

2. The operations have been adjusted to meet the strength of the patient, sometimes, beginning with the ligation of a single thyroid artery and progressing in stages to the removal of three-quarters or more of the enlarged gland.

3. Acute hyperplastic thyroids have given the most severe symptoms, but toxic adenomas have sometimes been almost as serious.

4. Adenomas and so-called colloid goiters have sometimes given distressing symptoms from pressure and unsightliness.

5. Some patients have improved under rest and medicinal treatment and the use of the *x*-ray, but so many failures have been noted in these forms of treatment that operation is believed to be the best form of treatment for a very large proportion of patients with goiter.

6. The pathology of cancer and growths which resemble cancer is very complex.

THE RESULTS OF IODIN ADMINISTRATION IN EXOPHTHALMIC GOITER

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In a paper presented before the Association of American Physicians in May, 1923, one of us (Plummer) gave a preliminary report on the results of the administration of the compound tincture of iodin (Lugol's solution) to patients having exophthalmic goiter, with the set of hypotheses which led him to the trial of iodin. A review of the literature on the subject will be given in other papers. The primary purpose of this paper is to present the effect of the administration of iodin on the postoperative mortality.

A brief partial resume of the theories and observations which led to the trial of iodin, and a correlation of these theories with the clinical complexes and hyperthyroid states, are first presented, as this is almost essential to classifying quickly the results that were anticipated and have been obtained from the administration of iodin. The hypotheses are briefly presented in a thematic way for the purpose stated, and hence do not fully represent our ideas.

The majority, at least, of goiters may be pathologically classified as diffuse colloid, adenomatous and diffuse hypertrophic and hyperplastic.

Adenomatous goiter, as a rule, has its inception in diffuse colloid goiter. In later years many of these goiters hyperfunction; that is, deliver sufficient of the product of the thyroid to the

organism to cause hyperthyroidism. The administration of iodin is apt to start this hyperfunction. The fear of giving iodin to patients having exophthalmic goiter rests erroneously on this observation and on the theory that an excess of iodin should augment a hyperthyroid state, as this element was considered and is now known to be a component part of the active agent of the thyroid. How its administration will influence the functional activity of adenomatous thyroids, once hyperfunction has started, is a debatable question that will not be discussed in this paper.

Evidence suggests that diffuse hypertrophy (and hyperplasia) of the thyroid develops as a result of an abnormally intense or continued stimulation of the thyroid, and that there are at least two conditions or groups of factors which will bring about an intensive stimulation of the thyroid: (1) Stimulation, originating in a hypothyroidism and acting through the normal mechanism on a thyroid whose ability to produce thyroxin is below the normal; this condition may be brought about by destruction of a large portion of the thyroid gland or an inadequate iodin supply—in either case the individual has a normal or deficient amount of thyroxin in the tissues. (2) Diffuse hypertrophy, produced by an intensive stimulation of unknown origin acting on a thyroid capable of normal response to stimulation. The latter theoretic concept explains the histologic findings in exophthalmic goiter.

In exophthalmic goiter the stimulus may at first be mild, causing hyperthyroidism before any notable histologic change takes place in the thyroid. Only rarely does such a case come to operation. When it does it is probably always, or at least with very rare exception, during a state of remission in the intensity of stimulation and not during a progressive onset. It is probable, if it has not been demonstrated, that the hypertrophy under a given degree of stimulation is less apt to develop, or if present is more apt to be regressive or disappear if there is an adequate iodin supply to the thyroid.

If a primarily normal thyroid is sufficiently stimulated exoph-

thalmic goiter will probably always develop. There is much to suggest that, as a rule, the stimulus of the thyroid in exophthalmic goiter acts on the gland as a whole and has its origin extrinsic to the gland; and that the stimulus in adenomatous goiter is largely intrinsic.

In 1912 Plummer pointed out that the physiologic status of patients having adenomatous goiters and those having diffusely hypertrophic thyroids is different; that only those having diffusely hypertrophic thyroids developed the complete complex of exophthalmic goiter; and that variations in the physiologic complexes correlated with the theory that two or three products, one or two of which give rise to the characteristics of exophthalmic goiter are driven from the thyroid.

In 1914 Kendall separated the active agent, thyroxin, 65 per cent of which by weight is composed of iodin. This immediately led to speculation as to the possibility of an incompletely iodized molecule being driven from the thyroid by intensive stimulation. An attempt to correlate this possibility with the varying complexes of the disease led to the following theories:

The clinico-physiologic complex of exophthalmic goiter varies with the total and relative amounts of completely and incompletely iodized dynamic agents in the organism. Many factors are conceived as possibly controlling the total and relative amounts of these agents in the body, as follows:

1. The well-known remissions and intermissions in the course of the disease point to variations in the degree of stimulation of the thyroid.

2. Training of the gland to function under abnormally intense stimulation leads to a better and larger product from a given degree of stimulation. This theory is in accord with well-known biologic laws and helps to explain a clinical complex and physiologic status that is apt to develop, as a rule, relatively late in the course of exophthalmic goiter.

3. The two preceding factors remaining constant, the amount of the abnormal product will within wide limits vary with the iodin available to the thyroid and possibly that available in the tissues of the body.

4. The physiologic status of the thyroid gland may at times be such that it can discharge suddenly a relatively large amount of the abnormal product.

Thyroxin is a catalytic dynamic agent hastening the rate of energy transformation in the body. It is a remarkably stable molecule that may continue its action for weeks before becoming exhausted. The evidence upon which the idea of an incompletely formed thyroxin agent rests also suggests that its life is short, acting much as adrenalin; in fact, such an agent may be responsible for findings that are confusing in the attempt to correlate the function of the thyroid and the adrenals. This agent may be directly responsible for the pigmentation in cases having exophthalmic goiter. It is also possible that experimental stimulation of the thyroid might drive this agent from the gland, and that it may be present in various extracts of the thyroid which have been introduced intravenously into experimental animals.

Many reactions that might follow the administration of iodin were considered. The complete iodization of the thyroxin molecule in the tissues seemed possible but not probable. That the iodin might lead to more complete iodization of thyroxin in the gland and that it might block its discharge seemed more probable. Many observations, beyond discussion here, suggested that an intensively stimulated thyroid is handicapped in its ability to pick up iodin from the circulation unless the concentration in the blood is high, and hence that large doses might tend to partially reverse the absorptive and discharge mechanism of the gland.

The primary clinical manifestations of the thyroid state associated with adenomatous goiter are, or are due to, the reactions that must accompany an elevated rate of energy transformation in the body: (1) Cardiovascular reactions that are indicative of an increased minute-volume flow of blood from the heart; (2) increased perspiration and surface temperature indicative of an elevation of heat elimination; (3) increased food consumption, in most instances insufficient to maintain the body weight; (4) a

group of findings attributable to stimulation, irritability and fatigue of the nervous system—not the characteristic nervous phenomena of exophthalmic goiter.

As we go deeper into the physiologic studies, the findings are all indicative of an increased mobilization of food and metabolites. Maintenance of the elevated rate of energy transformation above the normal ultimately results in the evidence of fatigue and degenerative changes in the tissues.

The clinico-physiologic complex of exophthalmic goiter can readily be conceived of as that following the administration of thyroxin, or that associated with hyperfunctioning adenomatous goiter plus certain notable characteristic findings that can be grouped as eye symptoms, the characteristic nervous phenomena, and the tendency to crises which may terminate in death. To these might be added a number of findings which, to avoid confusion, will not be considered.

For the purpose of this paper it is only necessary to divide the eye symptoms into exophthalmos and those that give rise to the peculiar "stare" present in about 65 per cent of the cases at the time of coming under observation. The stare is frequently present when there is no exophthalmos, and may disappear when a high degree of exophthalmos and hyperthyroidism is present. Prolongation of the physiologic status that gives rise to the stare probably causes protrusion of the eyeball. The stare fluctuates directly with the other phenomena attributed to the abnormal agent.

The entire nervous system seems to be in a status of hyperirritability and fatigue. The patient is restless, easily irritated and subject to emotional outbreaks. Intoxication is in many ways comparable to acute and chronic alcoholism. The lack of assurance that is so characteristic of psychoneurosis is strikingly absent if the patient is not in an emotional mood, characteristically depressed or fundamentally of a neurotic temperament. The patient frequently complains of an "inward" nervousness, which is the cause of what we are pleased to term "purposeful but useless motions." These are sufficiently characteristic to

permit of a diagnosis of exophthalmic goiter on inspection in at least 60 per cent of the cases. In rapid succession the legs may be crossed and uncrossed, the tie adjusted, a garment buttoned and unbuttoned, the nose wiped and so forth, almost without end. This series of motions may be interspersed with various poses. If sitting on an examining table the arms are apt to be thrown back and the hands rested on the table in such a way as to act as a crutch to support the exhausted muscles of the back. The motions are in a way an index of similar psychic reactions. A somewhat comparable status exists in the sympathetic nervous system, which on clinical analysis cannot be dissociated from the higher psychic reactions. If the status suddenly disappears the patient, in response to a question as to his condition, replies that he has passed into a state of complete repose or makes some similar answer.

In addition to the adjustment of the circulation, heat elimination, and so forth necessitated by the elevation of the basal metabolism, which as a rule seems to take place in a well-balanced way in the hyperthyroid state associated with the hyperfunctioning adenomatous goiter, there are evidences of chaotic attempts on the part of the sympathetic nervous system to maintain a balance in its coördinating function in the metabolic processes.

As the degree of intoxication becomes higher and the nervous phenomena become more marked, the patient is apt to thrash about on the bed and develop erythematous elbows. We have become accustomed to refer to such patients as being on the verge of a crisis. Vomiting, delirium and coma not infrequently develop in patients living in the Mississippi Valley. Sudden deaths are not infrequent. Deaths may be precipitated by seemingly slight causes, such as the hot-water injection into the thyroid or ligation of a superior thyroid artery. These deaths will be referred to as "crisis deaths." The nervous phenomena, eye findings and crises never develop in the course of hyperfunctioning adenomatous goiter.

To analyze accurately all deaths occurring in exophthalmic

goiter is impossible. The following generalizations, as suggested from an analysis of the deaths occurring in Rochester, previous to the use of iodin, may be made: The majority of non-operative and operative deaths, occurring under our observation, are divided between those occurring in crisis and those from definite infectious processes, such as pneumonia, endocarditis, suppurative cholecystitis, pyelonephritis and so forth. The relative number of the two groups has varied some from year to year. The study of the relationship of the development of the infectious processes to the exophthalmic goiter complex clearly indicates that latent infections become active when a crisis or near crisis is present, whether this develops spontaneously or is the result of an operation.

A relatively small percentage, approximately 1 per cent cannot be included in the crisis and infectious groups just discussed. This smaller group includes such causes of death as might be intercurrent in other than thyroid diseases: Cases in which damage to the laryngeal nerves is an important factor, and, as a rule, those cases having high basal metabolic rates in which the entire complex suggests that the thyroid is delivering little if any of an abnormal product. Such cases may die from intensive metabolic reactions that are hard to distinguish from the typical so-called crisis death. The same type of death is occasionally noted in cases having hyperfunctioning adenomatous goiter. When considered *en masse* these deaths seem to stand out quite distinctly from the typical crisis death, although it is hard to point out any definite difference in individual cases. The typical crisis never occurs in cases having hyperfunctioning adenomatous goiter.

The mortality percentages given in this paper, unless otherwise indicated, are obtained by dividing the number of deaths in any given period by the number of cases having their first goiter operation, in one of the hospitals associated with the Mayo Clinic, in the period. This gives a higher mortality than if patients who have had primary operations in previous years are included in the divisor, and affords a fairly accurate index

of the risk associated with the surgical treatment of exophthalmic goiter. All cases dying in the hospital following hot-water injections, ligations and resection of the thyroid are included in the operative mortality. In the non-operative mortality are included all other cases having exophthalmic goiter, dying in the city of Rochester, irrespective of the immediate cause of death.

The operative mortality rate for the ten years preceding the introduction of the use of iodin has averaged about 3.5 per cent. It has varied but little from year to year except to rise, as during the influenza epidemics.

The number of non-operative deaths for the years 1918–1922 were respectively 16, 18, 15, 10 and 16. During this period the operative mortality in the group of cases coming from that portion of the United States east of a line drawn north and south through the state of Indiana was less than 1 per cent.

An analysis of the mortality up to the time we began the use of iodin made any very material reduction in mortality seemingly impossible unless some radically new departure could be made in the management of the cases. An analysis of the deaths indicated that nearly 3 per cent of the mortality occurred under conditions such that death could not be attributed to errors in operative technic or to any methods of preoperative management, with which we were acquainted, except waiting for weeks and in some cases for months for the crisis status to disappear—this course would have increased the non-operative mortality and probably reduced the operative mortality.

The observations and hypotheses of the preceding portion of this paper were formulated long before the administration of iodin was started (in March, 1922); it, however, at first seemed wholly improbable that marked beneficial results, clearly demonstrable, could have been overlooked by previous observers. It was only when it became clear that, if the hypotheses were correct, one had but little chance of persisting in the administration of iodin to a solution of the question without clear correlation of the data on which the hypotheses rested that the

administration of iodin was started. The results obtained can be stated by enumerating the results that the hypotheses indicate should be obtained if iodin stopped or nearly stopped the delivery of the hypothetical abnormal agent: (1) The disappearance of the "stare;" (2) the disappearance of the characteristic nervous phenomena, including the crisis and crisis-death; (3) cessation in the development of exophthalmos and in some cases rapid regression; (4) reduction of the entire physiologic status to that of hyperfunctioning adenomatous goiter; (5) reduction of the symptom-complex to that of hyperfunctioning adenomatous goiter except for the anatomical findings in the thyroid, the exophthalmos, and minor physical findings which may remain, in a sense, an index of a preëxisting status—unless the patient is too near a moribund state before the iodin is started; (6) marked reduction of the non-operative mortality; (7) reduction of the operative mortality to less than 1 per cent; (8) reduction of operative mortality to a percentage approximately that of hyperfunctioning adenomatous goiter; (9) the reduction of the operative mortality to approximately that of the group of cases coming from that portion of the United States east of a line drawn north and south through the state of Indiana, before iodin was used; (10) absolute abolition of the mortality, both non-operative and operative, insofar as it is attributable to the crisis status and the infectious processes originating in the crisis, providing the iodin is administered sufficiently early in the disease.

The results obtained suggest several questions: Can the characteristic eye findings, nervous phenomena and crises develop in cases taking somewhere from 10 to 40 drops of Lugol's solution daily, or, stated in the terms of the hypotheses, will iodin absolutely prevent delivery of the abnormal agent? The evidence strongly suggests an affirmative answer (Plummer). If this is true many critics will, because of the many complicated factors involved, be slow to recognize the fact. Will the continuous administration of iodin over a prolonged period result in the delivery of a larger amount of the thyroid

agent, and hence to a higher degree of hyperthyroidism that would otherwise occur? Perhaps this should, on theoretic grounds, be anticipated; however, we have obtained little, if any, evidence to suggest such a result.

It is quite typical of a case of exophthalmic goiter to progress to the verge of a crisis or to a crisis and after a slight remission to regress to a much higher degree of hyperthyroidism, as indicated by basal metabolic-rate estimations. In the crisis the basal metabolic rate may be +30, or even less; later the basal metabolism may be +80 or higher, the patient gain weight, the stare go, marked exophthalmos be present and the entire complex indicate a well-trained gland delivering a normal product. In the latter condition another exacerbation of stimulation will cause a crisis, not infrequently followed by a higher basal metabolism and an apparent improvement in the patient's condition. The administration of iodin to a patient spontaneously pursuing such a course is apt to lead to the erroneous deduction that iodin temporarily benefits and then does harm. This course of exophthalmic goiter has very much confused the interpretation of therapeutic attempts in the past. The psychologic reactions of a patient with exophthalmic goiter and of all his friends are such as to bring the patient to the physician or a new physician at or near the height of the exacerbation, and whatever therapeutic measures instituted have been given an unwarranted amount of credit for the subsequent course of the disease. Many times the disease is on the whole progressive when everyone is enthusiastic about the results.

The only disagreeable results we have obtained from the administration of iodin have been an occasional rash, temporary swelling of the parotid gland and the complaint of tasting the iodin but rarely vomiting it. The former two conditions have been rare and caused but little annoyance; the latter is almost entirely avoided by administering the Lugol's solution in grape-juice or milk.

We began the use of iodin in more or less selected cases, giving 10 drops twice or three times daily. Later we changed

to 10 drops daily and found that in some cases this was not sufficient. Some cases made spectacular recoveries and were made absolutely well, as far as we could detect, when taking 10 drops of Lugol's solution daily, and were absolutely unaffected by 5 drops of Lugol's solution daily. Many of the selected cases were held at rest in bed for a week or so, in an attempt to avoid confusing improvement from rest, and the drop in basal metabolism due to rest, with the results of iodin administration. During the first few months it was given up to the morning of operation and then stopped. The surgical mortality for the year 1922 was reduced about 1 per cent and the non-operative mortality was as high as in preceding years. An analysis of the year's work and the results of iodin administration clearly indicated that, but for the use of iodin, the mortality would have been up to or above the average of preceding years. It seemed quite clear that the non-operative mortality would have been materially reduced, and the operative mortality reduced to approximately 1 per cent, had sufficient iodin been given routinely to all cases from the day of admission until any postoperative reaction, if present, had passed off. It seemed apparent that the majority of non-operative deaths had occurred in cases entering in a crisis or with infectious processes originating in a past crisis or a near-crisis status. This led to the immediate administration of 50 minims of Lugol's solution to all patients entering the hospital in crisis or with infectious processes present, and the giving of 10 minims of Lugol's solution twice or three times daily for at least seven days previous to operation and continuing this throughout the postoperative reaction. Patients in a crisis have, as a rule, been given 50 minims of Lugol's solution on the second day. Since this program was started there have been 2 non-operative deaths in crisis and 1 non-operative death from an infectious process. The 2 former entered in an apparently moribund condition and the latter in a seemingly hopeless condition. These patients probably would not have died had they arrived twenty-four hours earlier; the latter would probably have lived had iodin been administered during the crisis period in which the infectious process started.

Many cases have entered the hospital in extreme crisis, delirious or comatose, and many with beginning pneumonia and other infectious processes. Crises have always promptly disappeared except for the 2 cases noted above, vomiting when present has in most cases stopped within twelve to twenty-four hours; it has not in any case continued more than seventy-two hours. Recovery from infectious processes has been almost equally spectacular. A number of cases have come in in crisis and a developing bronchopneumonia. We have had no cases dragging along in crisis since starting the administration of iodin. The pneumonia as well as the crisis promptly disappears following the administration of iodin. Of course, this would not apply if we had happend to have had pneumonias that were not having their origin in the crisis status.

EXOPHTHALMIC GOITER. TOTAL MORTALITY, 1921-1924

	1921.	1922.	1923.	January 1 to September 1, 1924.
Cases operated on	561	491	564	600
Postoperative deaths	19	11	8	4
Non-operative deaths	10	16	6	0
	Per cent.	Per cent.	Per cent.	Per cent.
Surgical mortality	3.38	2.24	1.41	0.66
Ligation mortality	1.78	0.81	0.53	0.00
Total mortality	5.16	5.49	2.48	0.66

Since this paper was read the mortality statistics in the tabulation have been brought up to September 1, 1924. A complete review of the 12 operative deaths for 1923 and the first eight months of 1924 will be given in another paper.

The causes of all the operative deaths occurring in the first eight months of 1924 are quite clear: 2 from pneumonia, originating in technical difficulties; 1 from typical coronary sclerosis, and 1 from a hyperthyroid crisis. The case dying in a hyperthyroid crisis is particularly instructive. This patient had a single adenoma and little, if any, palpable thyroid. The symptom-complex at the time of entrance was that of hyperfunctioning

adenomatous goiter, and, although we did not know it at the time, Lugol's solution had been given for a considerable period up to one week before coming under observation. The administration of iodin had abolished the picture. During the week before the operation the patient was quiet and confined to bed for the greater part of the time. In this quiet condition the development of the nervous phenomena from the gradually increasing abnormal product were probably overlooked. The action of the iodin passes off quite precipitately at the end of five to eight days following cessation of administration.

DISCUSSION ON THE PAPERS OF DRS. TINKER, OCHSNER, DOWD, AND CHARLES H. MAYO AND HENRY S. PLUMMER

Dr. Alexis V. Moschcowitz, New York:

At the St. Louis meeting of the Association, I made a statement that there is very evidently a great difference in the treatment of exophthalmic goiter, which apparently depends upon geographical distribution. Thus, for instance, in New York City, and more especially at Mt. Sinai Hospital, exophthalmic goiter is very distinctively a medical disease. I was of the opinion that this preponderance in favor of medical treatment in New York City was due to carelessness and reluctance of the internists, who naturally see these cases first. A careful survey of the subject, augmented by manifold conversations with my medical confrères, led me to the belief that the fault lies deeper and is traceable to the surgeon. The internist has become more reserved because of the many failures he had with cases that were operated upon, and the reason for it was because cures after surgical intervention were reported which after a longer period of observation proved to be complete failures.

My own observation has led me to believe that the improvement which follows ligation of a single or even of two vessels is so fleeting that it should not even be considered as a curative measure. The improvement after unilateral lobectomy lasts longer. I doubt, however, that it lasts longer than six months, and many cases recur after this period; therefore, even this method cannot be considered curative. It is, however, an excellent temporary measure.

Finally we come to the most recent surgical method, viz., subtotal thyroidectomy. I have had excellent results with this method, and they have stood the test of time, in some instances for at least three years. Not one case has relapsed thus far.

I have come to the conclusion, therefore, that all the old statistics, *i. e.*, those antedating subtotal thyroidectomy, should be discarded completely and a new series started which includes the results of this operation only.

Dr. Edward P. Richardson, Boston, Mass.:

The use of iodin preliminary to operation, as advocated by Drs. Mayo and Plummer, seems to us at the Massachusetts General Hospital one of the most important advances that has been made in the surgical treatment of exophthalmic goiter. The effect of the drug has been studied at this hospital during the past year by Drs. Starr, Segal and

Means, who find a definite temporary benefit in about 80 per cent of the cases treated with it. In 40 per cent of the cases the drop in metabolic rate and pulse is approximately as sudden and marked as that following subtotal thyroidectomy. While the effect soon passes off, a period of remission in the disease is secured which is favorable for operation. From a surgical point of view the lessening of nervous tension and toxic reaction following operation is striking. The benefit to the patient and hospital can perhaps best be shown in the following way: Before using iodin, under a system of graded operations, in a small series of cases treated by the thyroid committee, the average number of operations per patient was about 2, and only 25 per cent of the cases had an immediate complete operation. In a few cases, since using iodin, the average number of operations per patient is about 1.3 and the percentage of immediate complete operations is about 80 per cent. Graded operations have proved their value and should not be abandoned, but I am convinced that they are less frequently necessary if iodin is used. The preliminary use of iodin makes it necessary to estimate on a new basis the risks and indications for operations.

Dr. Miles F. Porter, Fort Wayne, Ind.:

It is manifestly absurd to talk about permanently curing exophthalmic goiter by partial thyroidectomy. The only sure way to ensure against a return of the trouble would be to remove all of the gland, and, of course, that would not be wise. But when we leave enough of the gland *in situ* to carry on the physiologic function, we leave enough to render return of the trouble possible. I hardly know what is meant by "End-results in Goiter Operations." Really, one can only know the "end-result" in a given case after the patient is dead. I have known return of symptoms after ten and fifteen years.

I should like to emphasize what has been said about operating on old people. There is not a particle of sense in refusing a patient an operation because he happens to be sixty or eighty years old. Age in itself is no objection to operation, so far as I can see. If I had an enlarged prostate or a thyroid I wanted out, it would be up to the surgeon to give me my chance for my "white alley" regardless of my age.

Dr. Mayo (in closing):

This is an interesting series of papers. At the same time, if we are going to discuss results of operation, then the condition that produced the trouble in the beginning must be considered. There has been a

great deal of discussion throughout the world on the benefit of iodin in the prevention of exophthalmic goiter. The question comes up, Are we, as surgeons, doing our full duty to patients without discussing with them as to how they are to live or what they are to do to prevent a recurrence? About 10 per cent of these patients will relapse and about one-half of them will relapse enough to have a second operation, and one-half of these will again relapse sufficiently to require a third operation. It is not alone sufficient to say we have cured the patient if we do not do something to prevent a recurrence. From the surgeon's standpoint we have proved that patients have been benefited wonderfully by the use of iodine.

Dr. Ochsner (in closing):

There is no doubt in my mind but what the cures that we can obtain in operations for toxic and exophthalmic goiter depend upon two things: First, upon the condition in which the patient was at the time of the operation, and, second, upon a subtotal operation. In the cases in which all but 15 c.c. of tissue was removed we have practically no recurrence of the tumor, and of all cases operated on we have had a recurrence in only 5 per cent; consequently the argument of Dr. Porter does not hold good, because if you can prevent a recurrence of the tumor in 95 per cent by making subtotal excision, and, of course, ligating the vessels, I think that a great deal has been accomplished.

There is no doubt whatever but that the iodin treatment, which has come up again and again for the last three or four hundred years, has now come in a form in which it is useful.

A DISCUSSION OF THEORIES CONCERNING THE CAUSATION OF ACUTE AND CHRONIC PANCREATITIS

By EDWARD ARCHIBALD, M.D.
MONTREAL, CANADA

It is, I believe, three years since I brought this subject before the Association, and in the intervening time a certain amount of important experimental and clinical work has been published. I desire to report at this time a summary of the results of laboratory work done during the last two winters, and also to review generally the principal theories in the light of recent contributions to the literature. Let us take the latter point first, and begin with a brief statement of the current views.

There are three chief theories based on the supposed mode of entrance of the noxious agent into the pancreas: (1) The retrojection of bile into the pancreatic duct, as the result of a block at the papilla of Vater by stone (Opie), or by spasm of the sphincter of Oddi (Archibald); (2) the reflux of duodenal contents into the common duct and thence into the pancreas (Williams and Busch); (3) the spread of infection from the walls of the inflamed gall-bladder by way of lymphatics through lymph nodes, and on into the pancreatic stroma (Maugeret, Arnsperger, Deaver and Sweet). In the discussion of these various theories it is possible that insufficient distinction has been made between that form of pancreatitis which the surgeon is apt to call chronic (characterized by dull or subacute attacks of pain, and by the finding at operation of a distinctly enlarged and hardened pancreas, a change which Deaver maintains is usually confined to the head of the organ), and the acute form, which needs no particular description.

The argument has been advanced by Jones, of Boston, in a thoughtful and scholarly paper, that one must, in considering causation, distinguish between these two forms; and he believes that the chronic form frequently arises from a lymphatic spread of infection, while the acute is due to retrojection of bile.

For myself, as a working hypothesis, I prefer to assume that both chronic and acute forms are due to one cause, invasion of the pancreatic duct, by bile usually, or by duodenal contents sometimes, the difference in effect being due to differing grades of virulence. And I consider it experimentally proved that the injection of infected bile into the pancreatic duct, in varying amounts and of varying degrees of virulence, has produced, in cats, all the ordinarily known clinical varieties of pancreatic necrosis and inflammation from the mild chronic to the hyperacute. This has been accomplished both by direct injection with cannula placed in the pancreatic duct, and by running the infected bile through the gall-bladder under known pressures, in which case a closure of the common duct sphincter, presumably spasmodic, was the very probable assumption. On the other hand, attempts to get fluid to pass back into the common duct from the duodenum have failed; and the strength of the argument for this hypothesis is best found in clinical and autopsy observations, remarkably few in number, in which an area of acute pancreatitis, corresponding to the region drained by the lesser duct of Santorini, opening directly on the duodenal surface, has been found; and further by postmortem dissections of acute pancreatitis, in which the main pancreatic duct was found to open alongside but independent of the common duct in the duodenum. Of course, it is well known that the syringe injection of duodenal contents into the pancreatic duct is capable of causing acute pancreatic necrosis just as easily as does infected bile. But the theory still lacks direct experimental proof; that is, pancreatitis, so far as I know, has not yet been caused by such measures as tying off the duodenum below the papilla, or running infected fluids under high pressures into a closed sac of the duodenum. Nevertheless, it seems to be definitely proved

by postmortem observations that this theory is the only one that can account for a certain proportion of the cases.

The lymphatic theory, which would explain pancreatitis (to use Deaver's very apposite term) as a pancreatic lymphangitis, has gained in this continent very wide support in recent years, and particularly the support of the Mayo Clinic, as set forth in the papers by Judd and Mann, and by Mann and Giordano, based upon experimental and clinical observations. Graham's paper of last year also seemed to speak strongly for it. A number of other leading surgeons accept it, on the basis of clinical observations, as a good working hypothesis. Some of these articles, notably those of Mann and Giordano, and Deaver and Sweet, while bolstering up the lymphatic theory, have included an attack upon the theory of retrojection of bile by closure of the sphincter of Oddi; so that at present the chief interest—and let us say amusement—of the protagonists of these views and of their supporters lies in the controversy concerning them.

In what follows I propose to discuss the relative claims of these two theories upon the basis of the recent articles mentioned, particularly that of Mann and Giordano, and of Evarts Graham, and of our experimental work in McGill of the last two winters. The article of Mann and Giordano, which appeared in the *Archives of Surgery*, January, 1923, casts serious doubt upon the theory of the retrojection of bile. Their article is the result of a very large amount of experimental and clinical investigation and deserves close consideration. Lack of time prevents discussion of the whole of their paper, but one may at least try to answer their chief objections.

The first of these is an anatomical one. My assumption was that a spasmodic closure of the sphincter of Oddi might cause, and in animals did cause, a damming back of the bile into the pancreatic duct. For this it was necessary that the pancreatic duct should join the common duct at a point above the situation of the sphincter, which lies practically at the outlet. A number of investigators had shown by dissection that this

occurred in a fair proportion of cases, although there were variations in the percentage figures. Mann repeated these dissections in 200 cadavera, and concluded that such an anatomical condition of affairs was very much less frequent than was indicated by the figures of Opie, Baldwin, Ruge and others. He calculated that there was no more than a total of 3.5 per cent in which it would be anatomically possible for bile to be diverted in the way indicated into the pancreatic duct, and he therefore suggested that this rarity of the anatomical arrangement was a strong argument against the theory of bile retrojection. He went on to cite an examination of 11 cases of pancreatitis, presumably dissected at autopsy, in which the anatomy of the ducts, with regard to the situation of the sphincter, seemed to make it impossible that bile could have been forced into the pancreas. In these cases the two ducts opened side by side, or separately, into the duodenum, or the pancreatic duct joined the common so close to the papilla that retrojection was not possible.

To these arguments the answer is obvious. The contention of rarity on the anatomical side neither proves nor disproves anything. Pancreatitis in any case is a comparatively rare disease, and Mann's own percentage of 3.5 of anatomical conditions favorable to retrojection of bile is quite sufficient to account for the occasional case of acute pancreatic necrosis which comes to autopsy and in which the relation of the ducts can be demonstrated. We assume that his figures are correct, but we must not forget that they are much lower than those of other competent observers.[1] Moreover he did find one instance of pancreatitis in which dissection showed that bile had entered

[1] Since the reading of this paper there has appeared an article by Cameron and Noble (Jour. Am. Med. Assn., May, 1924, p. 1410), in which this problem is considered. These authors employed a new method of investigation. Producing an obstruction by impacting a small gall stone in the papilla of Vater, they forced bile or water, under very low pressures, down the common duct in seventy-four cadavera, and came to the conclusion that it is anatomically possible for a biliary calculus lodged in the ampulla of Vater to cause a reflux of bile up the duct of Wirsung in approximately 65 per cent of persons.

the pancreas. As to his series of 11 postmortem dissections, one may urge in rebuttal that in 3 recent postmortems on cases of acute pancreatitis occurring in Montreal the pancreatic duct in 2 joined the common duct well above the sphincter. At this point I might also say that my own dissections of the bile and pancreatic ducts in the cat disagree entirely with Mann's observations. In his well-known article upon the anatomy of the ducts in a large number of domestic animals, his figure illustrating the condition in the cat depicts the ducts opening into the duodenum side by side, whereas in a series of 10 cats, carefully dissected by Dr. Gibbons in our laboratory, and in a series of over 30 injections of the pancreatic duct through a cannula placed in it, it has been found that in all except 7 the pancreatic duct joins the common above the sphincter.

One may dispute, therefore, this argument on two grounds: (1) That Mann's dissections do not agree with those of others; and (2) even assuming their correctness, that the rarity of the favorable anatomical conditions may correspond quite well with the rarity of pancreatitis.

A second objection is again of an anatomical nature. In histological sections he has found certain muscle fibres (confirming in this the work of Helly), situated around the exit of the pancreatic duct, forming a sort of sphincter, and resembling, though much less well developed, the well-known sphincter of the common duct; and he observed that the two muscles were laid down in the shape of a figure of eight. He assumes that these must contract simultaneously, so that under such circumstances bile could not find its way into the pancreatic duct because the entrance would be barred by muscular contraction. To this one may reply that other observers have failed to find this pancreatic sphincter, or have found it in such small development that they deny to it any sphincteric action. Nevertheless, granting its existence, as the physiology of the liver and the pancreas and of their respective ducts are quite different, one having as to excretion a more or less continuous flow, while the other, the pancreas, has an intermittent flow during diges-

tion only, it is natural to assume that the sphincter muscles of their ducts do not as a rule contract simultaneously. In laboratory experiments a pancreatic fistula is seen to discharge juice only, or chiefly, during digestion, and then continuously, there being no evidence of sphincteric action; whereas with the common duct sphincter we see it opening and closing, letting out or shutting back the flow of bile. I cannot, therefore, agree with this argument.

A large part, however, of the article of Mann and Giordano concerned a repetition of our experiments in the production of pancreatitis in animals. Their conclusions, briefly, were these:

1. The possible pressure which the existing physiological mechanism (chiefly the gall-bladder musculature) can exert in order to inject bile into the pancreatic duct is relatively low. By this it is meant, I take it, that the *vis a tergo* is ordinarily too low to do this. The answer is that, while this may be true under normal conditions of physiological experiment, such observations cannot be assumed to hold good for pathologic conditions. The gall-bladder muscle may very possibly, under normal conditions, put forth no strong peristaltic action, but it is not proved that it cannot do so; and the comparatively large amount of muscle tissue in the gall-bladder wall suggests the possibility that it can contract strongly. On the other hand, any obstruction, spasmodic or otherwise, at the outlet of the common duct, if continued, as spasms elsewhere in the body are known to continue, for some length of time, may stimulate abnormal muscle contractions behind the point of spasm—witness the pain of colic. Moreover, it may produce a considerable collection of bile in the common duct behind it, and presumably a definite rise of pressure in that fluid. I have also called attention previously to a clinical observation of Dr. Starr's, which seemed to prove the presence of strong muscular action in the neck of the gall-bladder. Mann also has shown a rise of pressure in the common duct as high as 1000 mm. of water during vomiting, and he says that even sterile bile injected into the pancreatic duct at a pressure of 1000 mm. is capable of producing an acute pancreatitis.

2. Mann injected sterile bile into the pancreatic duct at the maximal pressure, short of that caused by vomiting, that could occur in the common bile duct, and found that this did not produce typical hemorrhagic pancreatitis, although definite damage of the pancreas sometimes occurred. One must point out that his experiments were all conducted with *sterile* bile, in which there was present, presumably, the normal amount of colloid material. It seems to me not really justified to make out of such observations an argument against the theory of bile retrojection in the human. The comparative innocuousness of clean bile containing its proper mucus, when forced into the pancreatic duct, was long ago established by Flexner, and has been confirmed by others. But I would recall the fact that five years ago I laid down, as one of the conditions for the production of acute pancreatitis by bile, that it should be an altered bile; and many experiments have proved that an infected bile produces extraordinarily greater damage in the pancreas than does clean bile. In a large proportion of cases of acute pancreatitis in the human, it is well known that the bile is altered, usually by infection, and the coincidence of infected bile passages with pancreatitis has often been emphasized. This argument, therefore, would seem not to apply.

3. In Mann's experiment the common duct in goats (a species in which the main pancreatic duct opens well up in the common duct) was ligated, thus forcing normal bile into the pancreatic duct. In these animals, numbering 5, no acute hemorrhagic pancreatitis followed, although in 3 the pancreatic ducts were found dilated and filled with bile-stained fluid. One should note, however, that in the 4 animals living longer than twenty-four hours there did occur well-marked changes in the way of atrophy of the gland, a connective-tissue proliferation, and even fat necrosis; in short, changes which might be expected to occur from the action of sterile bile. But one could almost guarantee that, if Dr. Mann had produced simultaneously an infection of the bile in the gall-bladder, or the bile ducts, there would have ensued an acute hemorrhagic pancreatitis.

A recent experiment, carried out some time after the reading of this paper, justifies this assumption. In a goat the common duct was tied below the entrance of the pancreatic duct, and simultaneously the gall-bladder wall was injected with a broth culture of Staphylococcus aureus; some also was thrown into the gall-bladder cavity. The result was an acute pancreatic lesion with extensive fat necrosis, and patchy parenchymatous necrosis.

In short, the experiments of Mann and Giordano do not imitate either the conditions usually found in the human, nor do they repeat my own experiments in animals. The fact that these authors used only sterile bile in their work renders their conclusions inapplicable to the lesion under discussion, that is, acute pancreatic necrosis, and vitiates their arguments in so far as they are intended to controvert the theory of bile retrojection. As to the causation of chronic pancreatitis, their experiments even afford some support to this theory.

To one statement of these authors I must especially refer. It is that in which they say it is impossible to get bile to pass into a pancreatic duct in most experiments at any pressure less than 500 mm. and that it is impossible to produce changes in the pancreas with bile introduced at pressures lower than 800 mm. Against this statement I would detail the following experiments:

Cat 243.—April 18, 1922: Ox-bile infected with Staphylococcus citreus was run into the gall-bladder under a pressure of from 300 to 600 mm. There occurred swelling, especially of the splenic end, to twice normal size; the organ became edematous and purplish red in color. On April 19 the animal was found dead. The pancreas at autopsy looked grayish and necrotic, with small hemorrhages. Slides showed necrosis everywhere. (Slide shown.)

Cat 286.—March 21, 1923: Ox-bile infected with Staphylococcus citreus was allowed to flow through a cannula inserted into the pancreatic duct at a pressure not above 370 mm.; 5 c.c.

were used. The pancreas immediately became much swollen and hemorrhagic throughout. A section was taken at operation 1 inch from the duodenal tip through a much congested area. The slides showed extensive necrosis of the lobules, very intense edema and a dense polymorphonuclear leukocytosis in vessels between necrotic lobules. The cat lived, but was killed two months later, when there was discovered an abscess in the pancreas and in the rest of the organ chronic edema with marked interstitial inflammation, and some replacement fibrosis. The original organism was recovered in cultures from the abscess. (Slide shown.)

CAT 293.—April 27, 1923: Ox-bile infected with Staphylococcus citreus on September 20, 1922, and kept at room temperature since, was run in by gravity at a pressure of 300 mm. in the manometer, through a cannula placed in the pancreatic duct. About 0.9 c.c. altogether entered the pancreas. Pressure not raised above 300 mm. In about twenty minutes after beginning the experiment it was found that the whole organ out to the splenic end was swollen to nearly twice normal size and was filled with a glassy edema, the vessels showing out strongly, much congested, but without obvious hemorrhage. The maximum effect was found in the area nearest the point of injection. On May 14, at 9 A.M., the cat was found dead. The entire pancreas at autopsy was seen to be converted into a dirty, grayish, necrotic mass, about normal size. The abdomen contained a large amount of thick, creamy and curdy yellowish pus. This was clearly due to a perforation of the duodenum at the site of the operative wound. A worm had apparently been caught in one of the silk sutures in the duodenal wall.

These experiments indicate that it is possible to produce acute pancreatic necrosis with infected bile entering the pancreas at a pressure of not over 300 mm. of water, a pressure decidedly less than that which the sphincter of Oddi is able to withstand. In these experiments, and in others, by using biles of varying

grades of virulence, such as the sterile bile of the particular cat itself, clean and infected bile from human cases of cholecystitis, infected ox-bile boiled and unboiled, or filtered, it has been possible to produce in the cat all those grades of pancreatic inflammation, with and without necrosis, which we are familiar with in the human. And in particular, as I pointed out at the Toronto meeting two years ago, that form of subacute and chronic swelling of the head of the gland, or of the whole of it, which Dr. Deaver claims especially for the lymphangitis theory, can be produced almost at will by the injection of bile which is only slightly infected, or with bile taken from a human case of subacute cholecystitis. These experiments will shortly be published in detail. And the histologic picture in both acute and chronic forms corresponds closely with that seen in human cases.

Considering the importance of the theory of lymphatic spread, so strongly advocated of late, particularly for the subacute and so-called chronic forms of pancreatitis, I felt it necessary to carry out experiments along this line. The work reported by Evarts Graham at last year's meeting, in which evidence was brought forward of some interstitial inflammation in the pancreas following injection of bacteria into a radicle of the portal vein, seemed at first sight to indicate a spread of infecton from the liver to the gall-bladder wall, thence by lymphatics to the chain of lymph glands between the hilus of the liver and the pancreas, and finally through these into the pancreatic stroma.

Barbour, of Brooklyn, has also reported, though with insufficient detail, some experimental work in favor of this theory. This is the only experimental work that I know of which might be construed as lending possible support to Deaver's theory.

During the past winter, Dr. Mark Kaufmann (working as the John McCrae scholar in the Laboratory of Experimental Medicine at McGill) has been busy with this subject. He will shortly report this work in detail, but I have his permission to record here a summary of his results. And first he repeated Dr. Graham's experiments. In 5 cats he injected a broth cul-

ture of Staphylococcus aureus and streptococci into a radicle of the portal vein. These all showed, it is true, slight inflammatory lesions in the pancreatic stroma, but they showed the same lesions in the substance of the kidney, heart, spleen, liver and wall of the gall-bladder; in 4 the organisms were recovered from the heart blood. (In the fifth the heart blood was not cultured.) Consequently, the result was simply a generalized blood infection, and we must conclude that the pancreatic lesions were not to be explained by a spread through lymphatics any more than were the similar lesions in kidneys, heart and spleen. Not content, however, with these negative or inconclusive results, Dr. Kaufmann, went on to establish a primary infection in the liver, in the gall-bladder and in the lymph gland lying upon the upper surface of the pancreas, in order to see if this infection might spread by lymphatics into the pancreas. In 4 cats the lymph gland just mentioned was injected with a few drops of a twenty-four-hour-old broth culture of Staphylococcus aureus. Of these 3 showed positive heart-blood cultures. In 3 there was inflammatory change in the adjacent portion of the pancreatic surface, in 1 amounting to abscess, but sections showed that the gland capsule had been broken through, and the extension of the inflammation was clearly by contiguity rather than by lymphatic spread. The inflammatory process did not extend into the rest of the organ, but was confined to the immediately adjacent area of the pancreatic capsule, and could be covered by two or three low-power fields of the microscope.

In 3 cats the same culture was injected under Glisson's capsule, at the edge of the liver near the edge of one of the right lobes. Of these 1 showed a positive heart-blood culture, and in this there were inflammatory changes in the spleen and in the gall-bladder wall. In 1 of these only was there inflammatory change, amounting to abscess, in the gland overlying the pancreas, and in this there was slight inflammatory change in the adjacent pancreatic capsule. In none of these was there any

change in the pancreas, except in the 1 with general sepsis throughout the body, and in that 1 the infection was confined to the surface of the pancreas at one point. (Section shown.)

In a series of 9 cats the cystic duct and the hepatic artery were ligated in 6; in 3 this was not done. In 6 the gall-bladder wall alone was infected with Staphylococcus aureus, while in 3 the lumen was injected in addition to the wall. All 9 showed a definite infection of the gall-bladder wall. In all 9 one or several glands of the hepatic chain of glands leading from the gall-bladder to the upper surface of the pancreas were the seats of definite catarrhal inflammatory change. In none was the pancreas grossly changed from normal. In 3 cases collections of small round cells were found in the stroma of the pancreas adjacent to the lymph glands, and also elsewhere in the pancreas, chiefly situated in the stroma. The changes were slight and purely microscopical. Very few such areas were found, and they were scattered. That they were definitely inflammatory is uncertain, because polymorphonuclear leukocytes were conspicuous by their absence. In these cases the gross appearance of the pancreas was normal. In the other cats that were killed within a short delay no such changes were found.

In 6 of the 9 the liver showed definite inflammatory change.

In 2 cases the pancreatic lymph gland was cultured and the organism found in 1.

Of the 9 cases only 1 showed a positive heart-blood culture.

In a further series of 2 cats the gall-bladder wall was injected in 1 with an iron solution and in 1 with methylene blue. Both the iron and the dye were demonstrated in the gland at the upper border of the pancreas, but not in the pancreas itself.

There would seem to be in these experiments some slight evidence in favor of a spread of infection, by the lymphatics, from the gall-bladder wall or the liver through lymph glands into the pancreas. But, after examining the microscopic slides myself, the inflammatory cells observed are comparatively so few in number, they are so regularly of the small round-cell

variety (which occurs normally in the pancreas), there is such a total absence of polymorphonuclears and of reaction on the part of the fixed connective-tissue stroma, and there is so little edema, in fact none that is definite, and so complete an absence of change in the gross appearance of the pancreas, that I cannot accept the evidence as proving the theory; in fact it is in my opinion strong evidence against that theory. One should note particularly that, in these experiments there was an entire absence of that swelling of the organ which we are accustomed to think of as being in the human the cause of attacks of pain.

The work of Bartels and that of Franke have frequently been cited by the supporters of the lymphatic theory. Their conclusions were based upon anatomical injections of Gerota's fluid into the lymphatics of the gall-bladder wall. The fluid was found to pass through or along side of the lymph node at the junction of the cystic with the common duct, and from there into the lymphatic nodes of the hepatic chain lying along the common duct, and toward the left into a node adjacent to the capsule of the head of the pancreas, and also one behind the duodenum where the common duct enters the duodenal wall. Bartel's conclusions were purely anatomical. He does not appear to have claimed that his injection extended beyond these glands into the pancreatic substance. Franke quotes his work briefly and repeated the injection in 14 cadavera. Going beyond Bartels, he applies his findings to the clinical problem, and concludes, without any addition of clinical evidence, that "The inflammation of these lymph vessels can lead ('führen können') in the sense assumed by Arnsperger, to a swelling of the pancreatic head." I would call attention to the fact that Franke's article appears simply to have been carelessly read by everybody. A careful examination of his article will show that his proof stops short of the pancreatic substance. Lymph vessels running over the pancreatic surface were occasionally found injected, but he gives no evidence of an injection of the pancreas itself. Dr. Kaufmann's work approached much more nearly

normal conditions in that he did not use the forcible injection method, but after placing methylene blue in the wall of the gall-bladder, and leaving Nature to transport the dye in the living animal, as opposed to the dead subject, he could find no evidence of the dye beyond the lymph node lying outside the pancreas. And in any case the purely anatomical argument of this theory disregards the laws of pathology.

The lymphatic theory remains unproved by experiment; on the contrary, experiment tends to disprove it. The bile-retrojection theory has still in its favor the bulk of experimental proof.

DISCUSSION

Dr. Fred B. Lund, Boston, Mass.:

The question of the cause of pancreatitis is of the greatest interest. It is hard to see how bile can enter the pancreas except in cases which show the peculiar anatomical arrangement of the ducts, so that a stone can block the opening of the common and pancreatic ducts and let the bile go back into the pancreas. Acute and chronic pancreatitis, however, occur without gall stones. The pancreatic secretions do not become active until activated by hormones in the intestinal contents. If they did the pancreas would digest itself. The pancreas does digest itself after death and very quickly softens. The pancreatic secretion is activated by the presence of necrotic tissue. Any sudden embolism may cause such necrosis and set self-digestion in motion, attended by hemorrhage with pancreatic apoplexy. The pancreas is a compound racemose gland, and there is a great deal of loose connective tissue extending between all the lobules of the gland, and there is also abundant connection between the lymphatic glands and vessels of the liver and of the pancreas.

In chronic pancreatitis we find swelling of the pancreas in which there is no evidence of blocking up of the bile ducts. How could bile be forced back into the pancreatic ducts when the absence of jaundice shows that it has not been forced back into the bile ducts themselves? It seems to me that Deaver and Sweet's work in this connection is very important. The fact that the most efficient method for the early treatment of pancreatitis is removal of the gall-bladder is evidence for the lymphatic and against the bile-regurgitation theory of pancreatitis.

In cases where the swollen pancreas has caused jaundice, however, the removal of the gall-bladder is the very worst thing we can do, as it prevents our use of it for the performance of a cholecystenterostomy, which is the only way in which we can get drainage of the bile. Even in the case of cancer of the pancreas with jaundice, cholecystenterostomy has in my hands given relief for long periods.

Another form of jaundice difficult to explain by the bile-regurgitation theory are the local abscesses which we sometimes find in the pancreas, which are cured by opening and drainage. It would seem difficult to explain in case there is regurgitation, because, as far as we can tell, it would extend entirely throughout its substance and would not produce localized abscesses.

I have seen cases of direct extension of pancreatitis or fat necrosis from an infected gall-bladder into the adjacent substance of the

pancreas, this taking place while we were waiting for a gall-bladder to quiet down before operation. Such a case as this seems to me excellent evidence for the infection by the lymphatic route.

Dr. Henry H. M. Lyle, New York:

I can recall a case that occurred on my service at St. Luke's Hospital which was operated on by one of the assistants. The diagnosis was made of an acute cholecystitis or acute gangrenous gall-bladder with perforation; at operation it proved to be acute hemorrhagic pancreatitis, with fat necrosis. The gall-bladder was drained and the patient made a satisfactory recovery until the sinus from the draining gall-bladder had healed. About two days after the healing of the sinus she was seized with another typical attack of acute hemorrhagic pancreatitis. The sinus was then reopened and thorough drainage established; with establishment of drainage the symptoms disappeared. The same phenomenon occurred again when the sinus was closed and the reopening alleviated the condition. This seems to be rather strong clinical evidence that there is some indirect relationship between the distention of the gall-bladder and the acute pancreatitis. As far as known the patient is now well.

Dr. Archibald (in closing):

I was familiar with Dr. Lund's views, and I know that the argument from the clinical side is apparently strong; but my plea is simply this, that until you can get some sort of experimental proof or clear clinical proof for the theory of spread by the lymphatics, it is perhaps a little premature to accept it as definitely as is being done. I see in the literature that there is a definite acceptance of that theory all over the country. It is remarkable how little proof there is of it; whereas, on the other hand, for the retrojection theory we have very strong proof, both clinically and experimentally, and we have also the proof that you can reproduce all grades of the disease in that way. Of course, I would not like to be understood as holding that the injection of bile is the cause in all cases, nor would I deny that in the future the lymphatic theory may be proved right. I can conceive that infection may overcome the gland barrier, invade the pancreas and produce an inflammatory response; but that certainly seems to me for the future to determine and for a lot of experimental work to decide. I am glad to hear of Dr. Lyle's case, offering, as it does, some clinical support for the retrojection theory.

ANEURYSM OF THE PALMAR ARCHES, WITH A REPORT OF AN ANEURYSM OF THE DEEP ARCH CURED BY EXCISION

By HENRY H. M. LYLE, M.D.
NEW YORK

The rarity of aneurysms of the palmar arches is surprising when we consider the vascularity and the great number of injuries to the hand.

In France, where more attention has been given to this subject, we find several excellent systematic studies. The American, English, Italian and German literature is confined to the reports of a few individual cases; the literature of the war, so rich in vascular injuries, has to date furnished but few examples.

We have been able to find 61 published cases. To this we are adding a personal case of an aneurysm of the deep arch, making a total of 62, 54 involving the superficial arch and 8 the deep arch.

Etiology. The general predisposing causes, which play such an important role in the formation of aneurysms in general, play but a minor part in the hand. This is due to the high incidence of trauma and the fact that more than half the cases occur in children and in young adults. There are a few reported cases of atheroma of the arteries and 2 of spontaneous formation. Insufficient data make it impossible to determine the relationship of syphilis.

The great majority of the aneurysms follow perforating or incised wounds by knives, nails, fragments of glass, etc. Contusions and occupational trauma are responsible for a few cases. It has followed cellulitis of the palm, and Roux reports a case following the reduction of a dislocated thumb.

Pathogenesis. The course of events is as follows: In the traumatic cases, after the external flow of blood has been checked, the escaping blood infiltrates the surrounding tissue and becomes organized and encysted. The false sac may or may not communicate with the bloodvessel; if it does we have a false aneurysm (arterial hematoma) or the extravasated blood is absorbed and a clot forms, sealing the wound in the bloodvessel wall. The clot organizes and becomes a cicatrix. This scar may give way or dilate under increased arterial strain. In the cases arising from external inflammation there is an actual pathological change produced in the external coat, which spreads to the essential middle coat, and this, becoming weakened, dilates before the arterial thrust. In the spontaneous cases there is a fatty degeneration of the muscle fibers of the middle coat and a granular degeneration of the elastic fibers.

Symptoms. The aneurysms vary in size from a pea to an apple, the average being the size of a small hazel-nut. The history is of a punctured or incised wound of the palm, which bleeds freely and in which the hemorrhage is difficult to check; occasionally there is nothing about the wound to make one suspect that he is dealing with anything other than an ordinary wound. A few cases have been caused by a single contusion, others by a series of repeated contusions received in the course of the patient's occupation. Within five to ten days after the receipt of the injury the patient notices a small pulsating tumor in the hand. The tumor has appeared as early as twelve hours and as late as five months. The original scar is often visible; the skin may or may not be discolored, sometimes it is ulcerated. The expansile pulsations are accompanied by a soft systolic murmur; a thrill is rare. The expansile pulsations are readily detected in true aneurysms; in the false the presence of clots may modify this symptom. The volume and expansibility of the tumor may be reduced or abolished by pressure on the brachial or by simultaneous pressure on the radial and ulnar arteries. The presence of sensory and motor symptoms can be explained by the relationship of the aneurysm to the palmar

nerve supply; they vary from slight weakness, tingling, numbness and anesthesia to excruciating pain and impotency of the hand. Over 80 per cent of the reported cases have had repeated hemorrhages.

Course. Abandoned to itself an aneurysm may lead to serious consequences; fortunately it is situated in such an exposed region that the patient is forced to seek medical advice and the tumor rarely attains a large size. Early surgical interference yields a prompt cure. Occasionally, pressure ulceration of the skin is produced. Suppuration and necrosis may result from the original wound or arise from compression and the application of styptics. There are no reported cases of aneurysm causing gangrene, but Keen reports a case of gangrene of the hand caused by injecting the sac with Monsell's solution, the gangrene necessitating an amputation at the wrist. Rupture with secondary hemorrhage is a frequent complication. Although few fatal cases have been reported, there are a number of cases in which the repeated hemorrhages have led to grave anemias. Spontaneous cure is possible, as evidenced by Verneuil and Taillaux's cases.

Diagnosis. The diagnosis in general is easy and is often facilitated by the history.

Synovial cysts, epidermal cysts, abscesses, lipomas, fibromas and sarcomas, due to transmitted pulsations, have been diagnosed as aneurysms. On the other hand, aneurysms have been incised under the impression that they were abscesses, vascular sarcomas, lipomas. Dupuytren operated for a lipoma of the hand and found an aneurysm. Verneuil operated for a fibroma and found the supposed fibroma to be an obliterated aneurysmal sac.

Treatment. The ideal treatment for aneurysm involving the superficial or deep palmar arch is excision. The incision of the sac after ligation of both ends of the artery has been practised with success. Schwartz considered excision of an aneurysm of the deep arch to be difficult, and contented himself with ligation of the radial and compression of the ulna. There are numerous

cases on record of failure after the simultaneous ligation of the radial and ulna, and if we stop to consider the various anomalies of the vascular supply of the hand it is evident that this unfortunate result could occur frequently.

Compression is uncertain and potentially dangerous, and should not be used except as a temporary expedient until surgical help can be obtained.

As a prophylactic measure, all narrow penetrating wounds that bleed freely and are in possible relation to the palmar arches should be explored and both ends of the artery secured.

FIG. 1.—Aneurysm of the deep palmar arch. Palmar view showing situation of the wound.

Exposure of the superficial arch is comparatively simple and needs no detailed description. For the exposure of the deep arch the choice of the incision depends on the situation of the aneurysm. We have used Delorme's internal palmar incision for the exposure of the deep arch in the hypothenar region and found it most satisfactory. Three of the incisions advocated by Delorme—the midpalmar, the internal palmar and the dorsal internal incision—are shown in Figs. 5, 6, and 7. The fourth, a dorsal incision, we have omitted, as it requires an excision of the upper part of the third metacarpal.

Traumatic Aneurysm of the Deep Palmar Arch. Male, aged twenty-six years, was admitted to the service of Dr. Lyle,

Fig. 2.—Aneurysm of the deep palmar arch. Shows the infiltration of surrounding tissue from the secondary hemorrhages and the aneurysm projecting through the wound.

at St. Luke's Hospital, May 8, 1923, referred by Dr. Dugdale. Family and past history immaterial. Present history: Four

Fig. 3.—Sac of aneurysm, 3.5 x 3 cm.

weeks ago he broke a glass bottle against a water faucet and sustained an incised wound of the right palm at the junction

of the thenar and hypothenar eminences. The wound bled profusely and sutures were required to check the hemorrhage. Eight days later the wound broke open and bled; ten days later a profuse hemorrhage took place; one week later there was a mild hemorrhage.

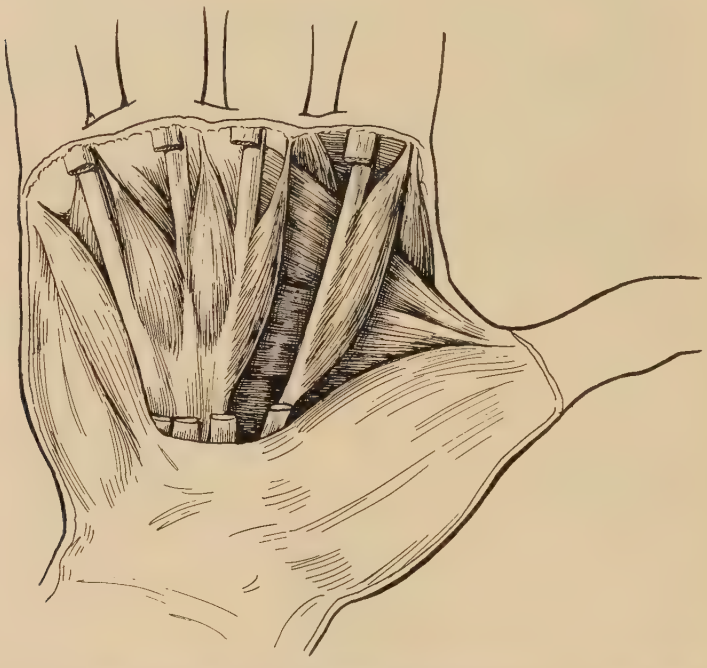

FIG. 4.—Shows the insertion of the palmar lumbricales on the deep tendons. Note the arrangement on the index. Delorme takes advantage of this to enter the palm here. Also note the adherent mass of tendons and lumbricales of the little, ring and middle fingers. Direct access to the deep arch is hindered by this mass, and in order to get a good exposure a retractor must be insinuated under this mass and the tendons retracted internally.

Physical Examination. Healthy young adult; Wassermann, negative. In the right palm, just below the junction of the thenar and hypothenar regions, there is an open wound 2.5 x 2 cm. The edges are everted and the surrounding soft parts are infil-

trated with blood. A pulsating swelling, about the size of a small hazel-nut, projects from the wound. The pulsations are

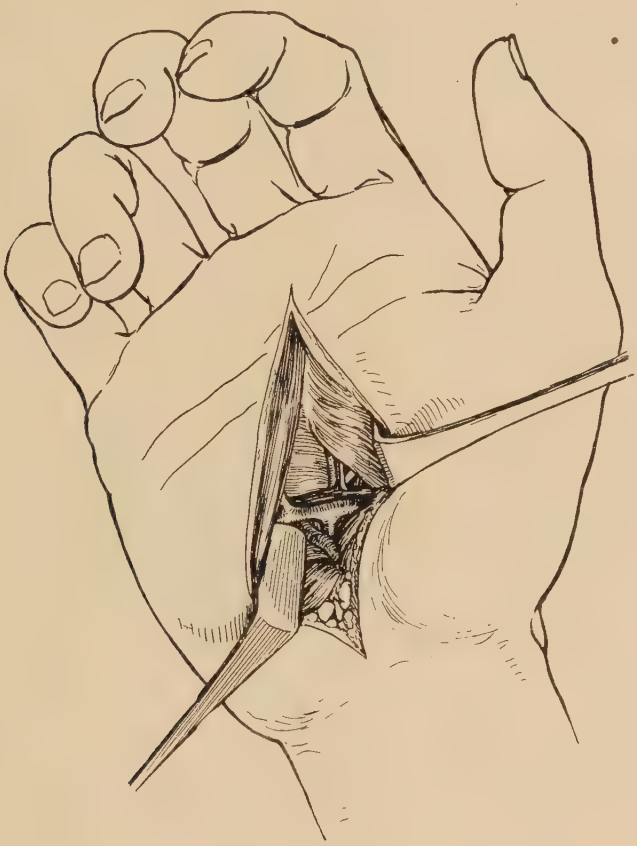

FIG. 5.—Shows midpalmar incision for exposure of the deep arch. The index tendon with its lumbricale is retracted externally; the mass of flexors with their lumbricales are retracted internally. This is facilitated by flexing the four fingers. The arch crosses the upper angle of the wound and is readily traced throughout. The motor branch of the ulnar nerve passes in front or behind the arch at the level of the 3d metacarpus.

expansile in character and are accompanied by a systolic murmur. Pressure on the radial and the ulnar arteries diminishes the

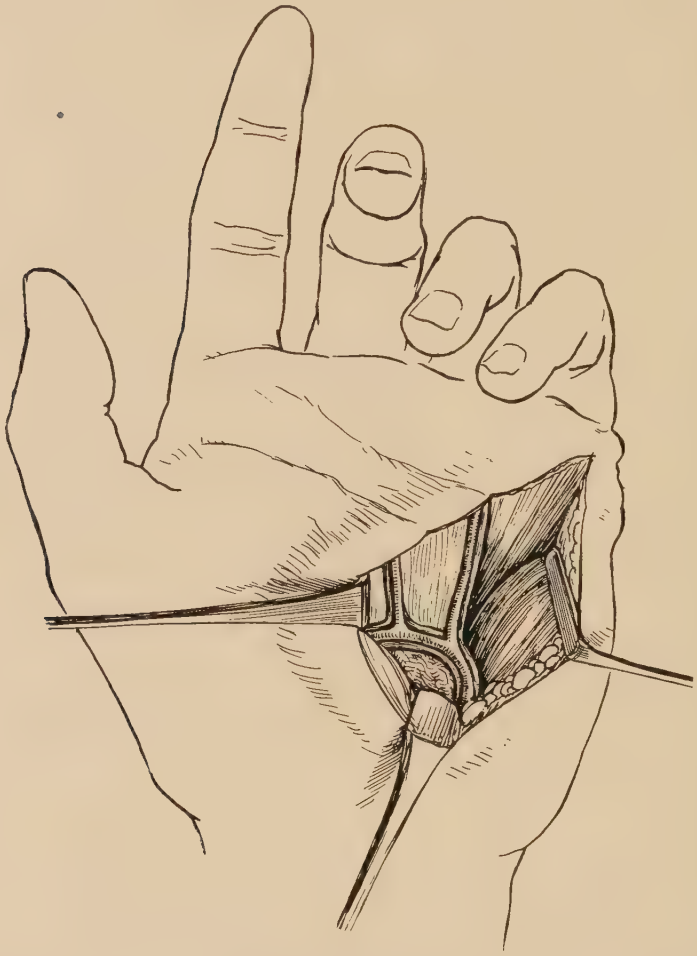

FIG. 6.—Internal palmar incision for exposure of the deep arch. The object is to enter between the flexor tendons and the external border of the hypothenar muscles. The flexor tendons with their tendon sheaths are retracted to the radial side, the hypothenar muscles and collateral nerves to the ulnar side. This is facilitated by flexing the 4th and 5th fingers. The arch is found in the superior portion of the wound and can be readily exposed to the adductor of the thumb. The ulnar nerve to the muscles of the thumb crosses sometimes in front, sometimes behind; this crossing takes place at the 3d metacarpus.

swelling, but does not obliterate it. The patient complains bitterly of pain in the little and ring fingers; both these fingers

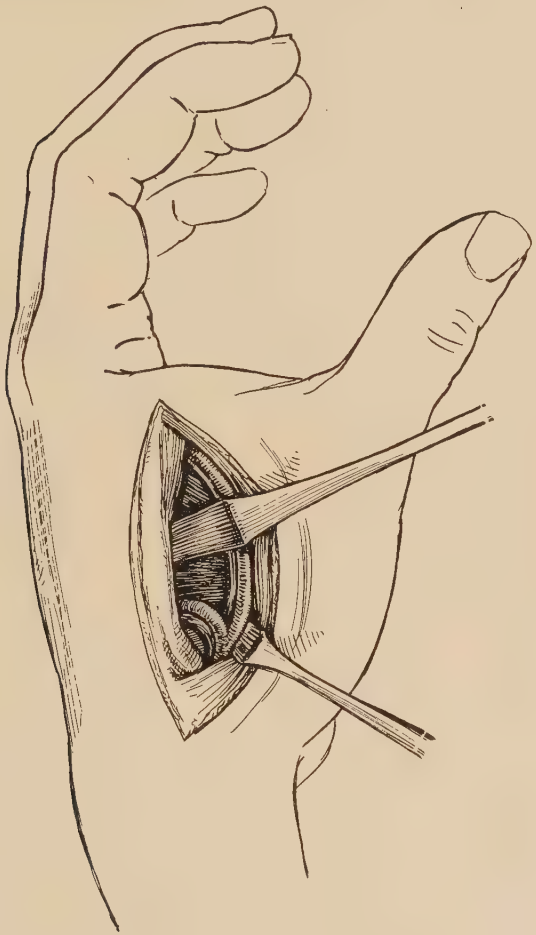

FIG. 7.—Dorsal internal incision for exposure of the radial origin of the deep palmar arch; this also gives access to the deep collateral of the index and thumb.

are flexed; the thumb is slightly adducted. The repeated hemorrhages have made the patient very apprehensive.

Name.	Age, sex.	Size, cause, location.	Time of appearance.	Notes.	Treatment.	Result.
Abéille, 1877	..	Half size of hazel-nut; cutting instrument; thenar branch of superficial palmar arch	15 days	Hemorrhages	Compression of radial	Cured.
Annandale	41 F.	Marble, sharp hook, ulnar side of ring finger	2 weeks	Annandale remarks that the proper way to treat such a case would be to open the sac, tie both ends of the vessel	Pressure applied for weeks with the effect of diminishing pulsation, but patient became restless and gave up the treatment	Failure.
Baumgarten, 1920	70 M.	Knife; thenar; left deep palmar arch	4 days	This is the 5th published case of aneurysm of deep palmar arch; diffuse hematoma; severe pain	Ligature of ulnar, palmar arch and accessory branches	Cured.
Bellamy	23 M.	Glass; hypothenar; superficial palmar arch	14 days	Repeated hemorrhage	Incision and ligation	Cured.
Bouchacourt, 1855	20 F.	Knife	1 day	Hemorrhages	Compression, direct and indirect, failure; perchloride of iron, failure; ligature of radial and ulna	Cured.
Bulteau, 1881	8	Size of nut; glass; right hypothenar ulna, where it forms a superficial palmar arch	1 month	.....	Compression of ulna, failure; electrolysis, failure; ligation of ulna at wrist, recurrence; ligation of radial at wrist and destruction of sac with actual cautery	Cured.
Caddy, 1896	23 F.	Hazel-nut; glass; right superficial palmar arch	4 days	Pain only present on closing hand; not tender	Ligature of radial and ulna at wrist	Cured.
Chandelux, 1890	38 M.	Large nut; glass; inferior portion of 3d interosseous space	2 months	.....	Excision	Cured.
Dalché-Menaud, 1920	M.	Small; knife; right hypothenar	Few days	Paresthesia	Intermittent compression	Cured.
Davey, 1855	20 M.	Nut; glass; right superficial palmar arch	4 days	First noticed pain in ring finger	Compress direct and of radial and ulna; had slough from direct pressure	Cured.
Dervaux, 1907	65 M.	Egg; knife; 1st dorsal interosseous space	.....	At original injury an attempt was made to secure both ends, but one retracted wound was packed	Excision three months later	Cured.
Dittel, 1863	15 M.	Pea; knife; left palm; superficial arch	4 weeks	Sensory disturbances in ulna region after ligature	Ligature of radial and ulna 1½ inch above wrist; recurrence fifteen days later; 2d ligature of ulna 1 inch above first	Cured.

Dubar, 1897	26 M.	Shoemaker's knife; deep palmar arch	.....	.....	Successive ligatures of radial; last to deep palmar arch	Cured.
Dupuytren, 1834	5 M.	Fragment of crockery; superficial palmar arch	12 days	Hemorrhages	Actual cautery	Cured.
Duvernoy, 1870	50 M.	Hazel-nut; blacksmith; repeated contusions; right thenar superficial palmar arch	Gradual	Pain in hand	Direct compression, then compression of radial and ulna	Cured.
Griffiths, 1897	23 F.	Spontaneous, 1¼ x ½ inch; hypothenar; branch to superficial palmar	.....	Considered it a case of local endarteritis	Excision	Cured.
Guattani, 1772	45 M.	Coachman; chronic irritation of reins and whip; hypothenar superficial palmar arch	Gradual	Mistaken for abscess	Incised and packed with gauze	Cured.
Guerineau, 1847	M.	Fragment of porcelain; superficial palmar arch	8 days	Hemorrhage	Indirect compression; ligature of ulna; galvanopuncture; recurrence in 29 days; galvanopuncture	Cured.
Guernnonprez and Besson, 1902	26 M.	Small nut; knife superficial palmar arch	.....	.....	Excision 1½ months after accident	Cured.
Happel, 1881	..	Acorn; catfish fin; left thenar; superficial palmar arch	8 days	.....	Direct pressure, failure; rupture of sac; sac opened; clots removed; ligature on radial in such a way as to include sac	Cured.
Haywood, 1871	F.	Glass; left superficial palmar arch	.....	.....	Incised sac; ligature above and below; ligatures cut through, due to soft tissue; ligature of ulna	Cured.
Herrgott, 1860	..	Traumatic; superficial palmar arch	.....	.....	Compression, failure; double ligature	Cured.
Jahresberecht, 1881	7	Nut; glass; left hypothenar; superficial palmar	.....	Grave anemia from repeated hemorrhages	Compression, failure; excision cure	Cured.
Jones, 1867	29 F.	Large; hypothenar	4 weeks	Pain with impairment of movement of fingers	Compression of radial and ulna	Cured.
Jones, S., 1877	15 M.	Hazel-nut; glass; left palm	5 days	Recurrent hemorrhages	Compression, failure; ligature of brachial	Cured.
Keen, 1882	Child	Sharp pebble; superficial palmar arch	.....	Amputation of hand	Compression and injection of Monsell's solution; this was followed by gangrene of hand, which necessitated amputation at the wrist	
Keen-Gross, 1893	38 F.	Recurrent aneurysm 12 years after, right hypothenar	.....	.....	Ligature of radial and ulna by Keen; original operation by Gross 12 years before; Gross performed 3 operations before securing a cure; operation on aneurysm; ligature of ulna; ligature of brachial; original injury was bruise of hand husking corn; 5 years of age; operation by local physician	Cured.

Name.	Age, sex.	Size, cause, location.	Time of appearance.	Notes.	Treatment.	Result.
Kendirdzy-Reclus	35 M.	Cherry pit; blow; right hypothenar	3 months	Paresthesia	Excision	Cured.
Kirwin, 1921	5 F.	Pigeon's egg; glass right palm; superficial palmar arch	5 days	Thrill felt; ring and little fingers flexed in palm	Excision	Cured.
Lamas, 1922	31 M.	Butcher knife; hypothenar; superficial palmar arch	.....	.....	Extirpation of sac; operation was performed by Nin-Y-Silva	
Lannelongue, 1877	..	Deep palmar arch	.....	.....	Compression of radial and ulna for 24 hours	Cured.
Lannelongue, 1877	..	Superficial palmar arch	.....	Threatened rupture	Incision of sac with ligature	Cured.
Letenneur, 1875	19 M.	Glass; midpalmar superficial palmar	1 month	Repeated hemorrhages; severe pain	Compression, failure; compression, rupture of sac	Cured.
Lyle, H., 1923	26 M.	Hazel-nut; glass; palm; deep palmar arch	4 weeks	Pain and hemorrhage	Excision	Cured.
Marais, 1920	14 M.	Nail; right palm; superficial palmar arch	.....	Mistaken for abscess; repeated hemorrhage	Ligature of radiopalmar, ulna and collaterals mistaken for abscess and incised; repeated hemorrhages; true nature discovered three days later. Grave anemia required intravenous solution	Cured.
Marjolin, 1859	12 F.	Glass; superficial palmar arch	6 days	.....	Intermittent compression	Cured.
Marques-Marques, 1920	M.	Small egg; agricultural laborer; repeated contusions; right thenar; deep palmar arch	.....	Mistaken for solid tumor; painful; right thumb and index interferred with	Incised by mistake; ligature and extirpation of sac	Cured.
Matas-Bradburn, 1919	52 M.	Injured right palm with shovel. Filled hypothenar region	5 months	Patient markedly arteriosclerotic Wassermann positive	Operation by Dr. M. Bradburn. Restorative endoaneurysmorrhaphy	Cured.
Matas-Stone, 1917	76 M.	Size of egg, gored by bull in first interosseous space at entrance of radial to form palmar arch	Present for 17 years.	No trouble until recently when it grew rapidly	Operation by R. L. Stone, assisted by Matas. Restorative endoaneurysmorrhaphy suture of orifice with fine silk obliteration of sac with catgut	Cured.
Mauclaire, 1908	31 M.	Cherry pit; glass; right palm; superficial palmar arch	24 days	.....	Excision of sac	Cured.
Maydl, 1896	33 M.	Size of walnut; fork; right palm; deep palmar arch	.....	Rapid growth, rendering hand useless	Incised and packed; ligature of ulna, where it becomes the deep arch	Cured.

Maydl,	46 M.	Size of walnut; knife; 1st interosseous space	3 weeks	Maydl's aneurysm of rami dorsalis art. radialis dextra	Excision three weeks after accident	Cured.
Mazade, 1866	43 M.	Knife; left palm; superficial palmar arch	9 days	Abscess and repeated hemorrhage	Compression; chloride of zinc; cauterization with compression of brachial artery	Cured.
Mollière, 1885	19	File; right palm; superficial palmar arch	Several days	At first small, rapidly increased in size, reaching maximum on 8th day	Profuse hemorrhage; ligature of superficial palmar arch; was not known if both ends were tied; recurrence; excised; cured	Cured.
Morestin, 1905	43 M.	Small nut; spontaneous; left hypothenar and palm; superficial palmar arch	.....	Spontaneous; patient recalls no contusion or wound	Excision six weeks after onset; it was necessary to sacrifice a small cutaneous nerve, which was adherent to sac	Cured.
Nélaton, 1862	M.	Traumatic; superficial palmar arch	.....	.....	Chemical cauterization; chloride of zinc	Cured.
Newbolt, 1908 Parker* (see below)	32 M.	One inch in diameter; chisel; left palm; superficial palmar arch	2 months	Pain and numbness in index and ring fingers	Applied for treatment two months after wound; incised sac wall and ligated superficialis volæ, ulna and communication to deep arch	Cured.
Parker, 1852	19 F.	Marble; glass; left superficial arch	10 days	Secondary hemorrhages; fingers flexed in palm	Cured by compression of brachial artery. This took sixty-four days and is a typical record of the pain and discomfort this method entails	Cured.
Pozzi, 1881	55 M.	Pigeon's egg; glass; left hypothenar; superficial palmar arch	8 days	Atheroma of arteries; severe pain	Direct and indirect compression; failure; incision of sac and ligature of both ends of superficial arch and the digital artery entering sac	Cured.
Rastouil,	26 M.	3 x 2 cm.; contusion; superficial palmar arch	8 days	No wound; loss of power in hand; sensory disturbances marked	Excision	Cured.
Regnault, 1912	37 M.	Pea; contusion; left superficial palmar arch	3 weeks	Naval gunner; contusions striking breech block to loosen it; no lues	Excision of sac	Cured.
Richet, 1882	M.	Revolver ball; superficial palmar arch	.....	.....	Rupture during treatment; compression	Cured.
Robertson, 1897	M.	Contusions; right hypothenar superficial palmar arch	5 months	Pain and numbness; was an engineer; blow on hand against engine lever	Compression, failure; ulna ligated at wrist	Cured.
Robinson,	35 M.	Hickory nut; struck hand to loosen tap; deep palmar arch	1 month	Possible lues 15 years before	Excision	Cured.

Name.	Age, sex.	Size, cause, location.	Time of appearance.	Notes.	Treatment.	Result.
Roux, 1837	25 M.	Apple; sustained it, reducing his own dislocation of thumb; right thenar	1 day	.....	This patient fell, dislocated, thumb and pulled it in place himself; next morning large swelling; incised hemorrhage; compression of radial and ulna, failure; later actual cautery; two months later came to Roux, who tied radial; proposed to tie ulna, hemorrhage; patient died before ulna could be tied	Died.
Salmon, 1920	25 M.	Large nut; knife; 1st interosseous space	.....	.....	Excision	Cured.
Schwartz, 1890	M.	Knife; deep palmar arch	.....	.....	Ligature of radial and ulna one month after accident	Cured.
Taillaux and Millet, 1891	10 M.	Cherry pit; glass; superficial palmar arch	8 days	Spontaneous obliteration	Direct compression with compression of brachial failure; large secondary hemorrhage; shock; on incision of sac it was found that a spontaneous cure had taken place	Cured.
Tuffier	20 M.	Size of franc; glass; hypothenar; terminal branch of ulna	8 days	Pain; movement restricted	Excision	
Verneuil, 1859	32 M.	Glass; palm; superficial palmar arch	Several days	.....	Compression of radial and ulna, failure; digital compression of brachial, with forced extension of hand	Cured.
Verneuil, 1866	M.	Shoemaker's knife; superficial palmar arch	5 days	.....	Compression and application of perchloride of iron, failure; incision and ligature	Cured.
Verneuil, 1874	..	Phlegmon of palm; superficial palmar arch	.....	Mistaken for abscess	Indirect compression, failure; injection of perchloride of iron; after multiple incisions for abscess of palm; secondary hemorrhages developed and later an aneurysm appeared. Verneuil states that arch was not injured by incisions	
Verstroete, 1902	24 M.	Hypothenar	.....	.....	Incision	Cured.
West, 1856	17 F.	Glass; left; between thumb and forefinger	10 days	Hemorrhages	Compression of radial and ulna; tourniquet to brachial	
Zynen, Vander, 1839	..	Wound of palm; left; spurious aneurysm	.....	.....	Cured by creosote	Cured.

Operation, May 10, 1923. Excision of aneurysmal sac. An open wound being present, a strict Carrel treatment was carried out for two days as a preparatory measure. Delorme's internal incision for exposure of the deep arch was used and gave an excellent exposure. The two main arteries, with collateral branches, were ligated and the sac excised. A wick drain was inserted and the wound closed. The pain ceased immediately. The wound healed in eight days and complete function was restored in four weeks. The patient has remained well. The false sac contained a hollow, laminated clot, 3.5 x 3 cm. in size, and was connected with the deep palmar arch.

Abstract of the Reported Cases. We include aneurysms of the superficial and deep palmar arches and their collaterals, also certain cases in which the trauma has been inflicted in the first dorsal interosseous space. We exclude cirsoid aneurysms, which are not uncommon. We consider Roberts, Mengers and Zichy-Wolanarski cases as being cirsoids. We have also excluded the few rare cases of arteriovenous aneurysms of the hand.

Conclusions. 1. Aneurysms of the superficial palmar arch are not common; aneurysms of the deep palmar are extremely rare, only 8 having been recorded.

2. The majority of the cases are caused by trauma with direct injury to the vessel wall, an insignificant number result from local or general pathological disease of the vessel wall, a small group are caused by chronic irritation and repeated contusions. Two cases have been reported as being spontaneous.

3. The best treatment is an early excision of the aneurysm; this applies to both the superficial and deep arches.

4. In selected cases where there is a well-formed sac the Matas restorative endoaneurysmorrhaphy is indicated.

5. As a prophylactic measure, all narrow penetrating wounds which bleed freely, and are in possible relation to the palmar arches, should be explored and both ends of the artery secured.

6. Although these aneurysms do not occur frequently, we

should remember their possibility, and in the prognosis of a trauma to the palm reserve a place for them.

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ENDOANEURYSMORRHAPHY

PERSONAL EXPERIENCE IN TWENTY-ONE CASES

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It has been twenty years since Matas presented a new and very rational method of treating accessible aneurysms, with a report of 4 cases, and in his last communication[1] he states that 350 endoaneurysmorrhaphies have been reported, including 59 of his own. Though this figure does not, of course, represent all the cases operated upon by this method, it seems, nevertheless, too small a number, particularly when one considers the great number of traumatic aneurysms which must have resulted from wounds of the bloodvessels in the late war. From personal observation and verbal reports I know that many ligations were done for the cure of these aneurysms where endoaneurysmorrhaphy would have better served the purpose, in view of the fact that before the war enough cases had been reported to show that the Matas operation resulted in a lower mortality, a higher percentage of cures than the old ligation methods, and that it had practically eliminated gangrene, which, under the most favorable circumstances, followed the ligation of large arterial trunks in a certain percentage of cases.

Sencert, of Nancy, in his book[2] recommends extirpation rather than ligation in traumatic aneurysm, and it seems to have been the method preferred by most French surgeons during the war.

[1] Résultats immédiats et éloignés de la Cure des Anévrismes, La Presse Médicale, No. 10, du 3 Fevrier, 1923.

[2] Wounds of Vessels, University of London Press, Ltd., 1918.

He says that gangrene is less frequent after extirpation than after ligation. In 93 cases of extirpation, which he collected from French and foreign literature during the war, gangrene followed in 9 cases. In the British Service, ligation was the operation of choice. The Germans practised suture of the injured vessel rather than ligation more extensively than the Allies, and they also utilized the intrasaccular ligation (the passing of a suture through the sac wall and about the artery) in aneurysms where the restorative operation of Matas could not be done. Matas[1] says they used the restorative operation in about 50 per cent of the saccular aneurysms. Most of the German surgeons gave up the extirpation of the sac in favor of the above-mentioned methods. It is impossible here to discuss in more detail the wide difference of opinion held by surgeons of the different countries in regard to the treatment of traumatic aneurysms, but the whole question, including the immediate treatment of wounds of the vessels, is thoroughly discussed by Matas in Keen's supplementary volume, published in 1921. I am convinced that the principles laid down by him are applicable to traumatic as well as syphilitic aneurysms, and it is to be regretted that more endoaneurysmorrhaphies were not done during the war.

These observations have caused me to feel that an additional report of my personal experience may be timely and might prove the means of stimulating others to deal with aneurysms from within the sac and abandon ligation, which, though easier to do, carries with it a higher mortality and a smaller chance of cure.

I reported my first experience with the Matas operation in 1904[2] and my second in 1907,[3] and in 1912[4] I presented a summary of my experience in 8 cases. Since the publication of this paper I have operated upon 13 additional cases, bringing the total up to 21 cases. It might be added that no ligations for the

[1] Keen's Surgery, supplementary volume, 1921.
[2] American Medicine, August, 1905.
[3] Annals of Surgery, September, 1907.
[4] Jour. Am. Med. Assn., July 27, 1912.

cure of aneurysms have been done during the past twenty years, except for hemorrhage and except in aneurysm of small vessels, such as the temporal and radial. I have also done one successful invagination operation on a small traumatic aneurysm of the common carotid.

Of the total 21 Matas operations, I would like to exclude from the present discussion 1 case of aneurysm of the abdominal aorta, already reported, and 1 of the splenic artery, and consider only the remaining 19, all of which had to do with what are generally considered accessible aneurysms. Of these 19 there were 6 popliteal, 10 femoral, 2 femoral and external iliac and 1 brachial. Six were traumatic and 3 of these were arteriovenous.

Deaths. There were 3 deaths following operation; 1 was an anesthetic death just at the conclusion of the operation; 1 occurred fifteen days after operation and twenty-four hours after a ligation of the femoral artery for secondary hemorrhage from the site of an obliterated popliteal aneurysm. The patient was in good condition following ligation and died very suddenly the next day; the autopsy showed a vegetative endocarditis and splenic and renal infarction. The third death was in an extremely ill luetic negress with a femoral aneurysm. The operation was done under local anesthesia because of the patient's very grave cardiac condition. One week after the operation she had a hemorrhage from the site of operation, which was controlled by reopening and resuturing. She died suddenly the next day without any more bleeding or any symptoms indicating impending death. The autopsy findings in the case history, No. XV, I think, show several sufficient reasons for her death.

I probably ought to say that one of my early cases of popliteal endoaneurysmorrhaphy died of uremia two months after operation, when his wound was healed. This man, a luetic, had chronic Bright's disease at the time of operation and local anesthesia was employed. Therefore, it is not likely that the operation hastened his death.

In reviewing these deaths I do not think, with the exception of the second, that they can be attributed directly to the opera-

tion or that any different results would have followed ligation. Two of the 3 patients had far-advanced syphilis, lesions of the heart, aorta and viscera.

Hemorrhage. In addition to the cases above mentioned, secondary hemorrhage occurred in 2 others, both traumatic femoral aneurysms. In 1 the bleeding occurred two weeks after operation, when the wound was well healed. It was controlled by resuturing, but recurred four days later and a ligation of the femoral was done. The patient made a good recovery. In the second case, an enormous arteriovenous aneurysm, bleeding occurred three weeks after operation, due to late infection, which I am sure resulted from the injudicious insertion of a soft rubber drain two weeks after operation, because of a superficial collection of serum. Resuture was done in this case also, but bleeding recurred and ligation of the femoral was done. This patient made an excellent recovery.

It will be noted that the bleeding in all these cases occurred from one to three weeks after operation. I think that secondary ligation two or three weeks after operation carries with it practically no danger of gangrene, as the collateral circulation is by this time well established.

Gangrene, Amputation and Secondary Aneurysm. There has been no gangrene and no amputation in any of these cases, including the fatal ones, and in this connection I would call attention to the fact that 10 were aneurysms of the femoral and 2 of the femoral and external iliac. There has been no secondary aneurysm. These results should be compared with 10 per cent of gangrene in the 93 cases of extirpation of traumatic aneurysms reported by Sencert.

Results. The 16 cases which survived operation have nearly all been traced, as shown by the case reports, and none have had any recurrence or any circulatory disturbance.

Technic. It must be apparent that the treatment of aneurysms, resulting from disease of the bloodvessels, is different from that of those caused by direct injury of the vessel. In the case of the diseased vessel the patients are practically all syphi-

litics, have more or less advanced disease of the heart and aorta and often have liver and kidney lesions. In these cases far better results will be obtained if operation is preceded by an intensive antisyphilitic treatment, including large doses of the iodides. The pain and the growth of the aneurysm can be fairly well controlled by complete rest in bed and the constant application of an ice-bag over the aneurysm. It has been our rule to keep up the use of mercury and iodides after the operation. Some of our cases have easily taken 200 to 300 grains of potassium iodide a day. The improvement which takes place during this preliminary treatment has been quite remarkable in a few cases, and it certainly has reduced the operative risks.

In these cases, too, I would urge that the control of the circulation should only be made by digital compression. Temporary ligatures or rubber-covered artery clamps for the control of the vessel above the aneurysm are likely to so injure it as to cause the subsequent development of an aneurysm at the site where they have been applied. Such cases have been reported, just as they have after permanent ligation of the artery for the cure of aneurysm. For this reason, too, the tourniquet has not been used in our recent cases, although with it the risk of damage to the vessel is not so great. Digital compression of the vessel by an intelligent assistant is quite reliable, and has only to be kept up for the few minutes it takes to empty the sac, and to find and close the large openings into it. In the high femoral and the femoroiliac aneurysms the abdomen has been opened and the common iliac compressed by an assistant. These precautions are not necessary in the traumatic aneurysms, but even in these cases I think the temporary ligature should be avoided, though the tourniquet or rubber-covered clamps may be employed. Elevation of the extremity for a few minutes before the sac is laid open will do a good deal to conserve blood. In none of the cases here reported has the amount of blood lost at the operation been sufficient to affect the patient's general condition.

In all the aneurysms due to disease of the vessel I have done the obliterative operation. In most of the traumatic aneurysms

and in the three arteriovenous aneurysms the restorative type was done. I have not done the reconstructive operation and I have doubted its applicability, except in rare instances of traumatic aneurysm, where a large part of the vessel wall is normal. In 2 cases of arteriovenous fistula, 1 of the brachial and 1 of the femoral vessels, the opening in the artery was closed, the vein ligated and a portion of its wall used to reinforce the closure of the artery.

I have come to the conclusion that catgut is the best material to use for closing the openings into the sac and for its obliteration, although I used linen thread for the deeper sutures in my earlier cases.

I have never used the mattress sutures through the skin to aid in obliteration of the sac, as was suggested by Matas in his original paper, because I thought that a suture passing through the skin and into the sac might be the means of causing infection.

Infection is a serious complication and may readily lead to secondary hemorrhage. This was certainly true in one of our cases, an enormous arteriovenous aneurysm of the femoral vessels. One of the most important measures to take in order to avoid infection is complete hemostasis within the sac and its complete obliteration. Pockets where blood and serum accumulate are dangerous foci of infection. Another source of infection is any kind of a drain, even the subcutaneous type. In order to prevent any subcutaneous accumulation of serum it is better to leave considerable space between the skin sutures than to use a drain, even for twenty-four to forty-eight hours. If any superficial infection should occur the skin sutures should be promptly removed and no drain used. In the syphilitic cases I formally used a superficial drain, but considerable experience in war wounds has caused me to abandon the practice, as it is more apt to cause, than prevent, infection.

In arteriovenous fistula the artery and vein are always densely adherent for a considerable distance above and below the point of communication, and their separation is tedious and difficult. In these cases it seems better to ligate the vein below and above

the opening, without attempting separation from the artery, then to open the vein, with the arterial circulation controlled, and suture the opening into the artery. The vein wall between the ligatures can then be used to reinforce the arterial closure. In one of my cases a large tributary vein opened into the involved vein between the ligatures, and should this occur the tributary vein should be ligated beyond the main vein.

In the true arteriovenous aneurysm it is unnecessary, I think, in most cases, to dissect out the vessels above the aneurysm. A tourniquet should be applied, the sac opened and the necessary occlusion done from within the sac.

I am inclined to believe after this review of my personal experience that the Matas operation is indicated in all accessible aneurysms, and that it should supplant ligation, because its dangers are less and permanent cure is more assured.

The following is a brief synopsis of our 21 cases. The first 8 cases were reported in more detail in 1912:

Case I.—Negro, male, aged thirty-one years. Pennsylvania Hospital, operation September 29, 1904. Syphilitic. Popliteal aneurysm. Esmarch constrictor. Obliterative operation. No drain. Superficial infection. Eight years later patient was perfectly well.

Case II.—Physician, aged fifty-seven years. Bryn Mawr Hospital, operation November 24, 1906. Syphilitic. Popliteal aneurysm and advanced Bright's disease. Local anesthesia and a little ether. Esmarch constrictor. Obliterative operation. Complete obliteration impossible; packing used. Wound healed by granulation. Patient died two months after operation from uremia.

Case III.—Negro, male, aged thirty-eight years. Pennsylvania Hospital, operation November 27, 1907. Large femoral aneurysm. Probably not syphilitic. Digital compression of femoral after exposure. Obliterative operation. Superficial

drain. Slight infection. Discharged with healed wound January 4, 1908. Letter received five years later reported patient perfectly well.

Case IV.—White, male, aged forty-four years. Jefferson Hospital, operation December 7, 1907. Large traumatic femoral aneurysm. Esmarch constrictor. Obliterative operation. Patient died three years later from tuberculosis without any further trouble from the aneurysm.

Case V.—White, male, aged forty-five years. Jefferson Hospital, operation September 13, 1908. Aneurysm of abdominal aorta, complicated by anuria, which suggested an aneurysm of the renal artery. Symptoms suggestive of rupture on day previous to operation. Aorta controlled by rubber catheter and obliterative operation done. Patient died on the table.

Case VI.—White, male, aged forty-six years. Pennsylvania Hospital, operation January 11, 1911. Popliteal aneurysm. Esmarch constrictor. Obliterative operation. Superficial drain. Slight discharge from wound. Quite well eighteen months after operation.

Case VII.—White, male, aged thirty-six years. Jefferson Hospital, operation July 12, 1911. Aneurysm of femoral and external iliac (syphilitic). Abdomen opened and common iliac controlled by digital compression. Obliterative operation. Patient examined eight years after operation; excellent result; no symptoms.

Case VIII.—Negro, male, aged thirty-six years. Jefferson Hospital, operation June 1, 1912. Popliteal aneurysm (syphilitic). Esmarch constrictor. Obliterative operation. Anesthetic death as skin sutures were being introduced. Autopsy negative except aortic atheroma.

Case IX.—Negro, male, aged thirty-two years. Pennsylvania Hospital, operation November 8, 1916. Femoral aneurysm (syphilitic). Abdomen opened; digital compression of external iliac. Obliterative operation. Superficial infection. Discharged January 11, 1917; wounds healed.

Case X.—White, male, aged twenty-six years. Jefferson Hospital, operation January 6, 1917. Popliteal aneurysm (syphilitic). Confined to bed for six months. Digital compression of femoral without exposure. Local anesthesia and a little ether. Obliterative operation. Secondary hemorrhage. Ligation of femoral under local anesthesia two weeks after operation. Died suddenly next day. Autopsy findings: hypertrophy and dilatation of heart; fatty degeneration; acute vegetative endocarditis; splenic and renal infarction; red atrophy and fatty infiltration of liver and atheroma of aorta.

Case XI.—White, male, aged eleven years. Jefferson Hospital, operation April 3, 1917. Traumatic femoral aneurysm; three years' duration; gunshot wound. Digital compression of femoral without exposure. Restorative operation. Two weeks after operation, with wound healed without infection, bleeding began. Resuture and transfusion. Bleeding began four days later and femoral ligated under local anesthesia. Discharged with wound healed and pulsation in posterior tibial May 24, 1917. April 28, 1924, letter reports patient perfectly well; "cannot tell one leg from the other," and plays football.

Case XII.—White, male, aged twenty-six years. U. S. Base Hospital No. 19, London: November, 1918. Traumatic femoral aneurysm. Shrapnel. Restorative operation. Healed without infection. Evacuated to United States. Letter received April 19, 1924, reports no further trouble from aneurysm.

Case XIII.—White, male, aged twenty-six years. Pennsylvania Hospital, operation November 20, 1920. Arteriovenous

aneurysm of brachial vessels. Machine-gun bullet two years previous. Vein ligated above and below. Artery controlled by rubber-covered clamp compression. Restorative operation with reinforcement with section of vein. Discharged with wound healed. Unable to trace.

Case XIV.—Negro, male, aged forty-seven years. Pennsylvania Hospital, operation November 30, 1921. Popliteal aneurysm (syphilitic). Esmarch constrictor. Obliterative operation. Examined two years later; no further trouble from aneurysm, but has cardiorenal disease.

Case XV.—Negress, aged twenty-three years. Pennsylvania Hospital, operation December 21, 1921. Femoral aneurysm (syphilitic). Acute endocarditis; fever; delirium; symptoms of splenic infarction. Improvement under antisyphilitic treatment. Very bad operative risk. Local anesthesia. External iliac exposed. Digital compression. Obliterative operation. Bleeding seven days later. Resuture under gas anesthesia; no more bleeding. Died suddenly next day, when apparently in good condition. Autopsy findings; acute and chronic endocarditis and aortitis; hypertrophy and dilatation of left ventricle; infarction of spleen and kidneys; atrophic cirrhosis of liver and vesicular emphysema of lungs.

Case XVI.—White, male, aged twenty-seven years. Pennsylvania Hospital, operation September 27, 1922. Arteriovenous fistula of femoral vessels; gunshot wound twenty-six days previous. Esmarch constrictor. Restorative operation. Vein ligated and portion used to reinforce closure of artery. Prompt healing. Examined and found in good condition some weeks after discharge, but unable to trace since.

Case XVII.—White, male, aged thirty-five years. Jefferson Hospital, operation October 16, 1922. Enormous arteriovenous aneurysm of femoral vessels. Stab wound, with scissors, four

months previous. Esmarch constrictor. Restorative operation done on both vessels. Wound healed primarily. Two weeks later some serum evacuated and, without instruction, a drain inserted. Later infection and bleeding. Sac reopened and bleeding vein sutured. No bleeding from sites of previous suture. Wound packed open because of infection. Transfusion. Bleeding occurred at the end of week and again controlled by suture within the contracted sac. Another hemorrhage a few days later. Ligation of superficial femoral. Made good recovery and is now perfectly well.

Case XVIII.—Negro, male, aged thirty-five years. Pennsylvania Hospital, operation November 14, 1922. Popliteal aneurysm (syphilitic). Digital compression of femoral without exposure. Obliterative operation. Returned to work in post office two months after operation and was without symptoms and still working November 14, 1923, one year after operation.

Case XIX.—Negro, male, aged fifty-one years. Pennsylvania Hospital, operation January 9, 1923. Aneurysm of femoral and external iliac (syphilitic). Extended up to within two inches of the umbilicus. Abdomen opened and abdominal aorta controlled by digital compression. Obliterative operation. Discharged March 3, 1923, with wound completely healed. April 21, 1924, has had no further trouble.

Case XX.—White, male, aged fifty-four years. Jefferson Hospital, operation March 5, 1923. Aneurysm of splenic artery. Not (?) syphilitic. Preoperative diagnosis, upper abdominal tumor, probably retroperitoneal. Mass found attached to tail of pancreas, which was thought to be a cyst. No pulsation. During separation rupture occurred, with profuse bleeding and evacuation of clots; finger introduced into sac; discovered opening of vessel and bleeding was easily controlled while quantities of clot were evacuated. The arterial opening into the sac was closed by suture and the sac obliterated. Patient was in good

condition the next day, but then developed a bronchopneumonia and died on the third day, with a high temperature. He had no symptoms of bleeding and no abdominal symptoms. Autopsy findings: aneurysm of splenic artery; no blood in abdomen and no peritonitis; marked fibrosis of aorta; sclerosis and dilatation of left common iliac; acute suppurative bronchitis and early bronchopneumonia. Wassermann from heart blood negative.

Case XXI.—Negro, male, aged forty-five years. Jefferson Hospital, operation April 23, 1923. Femoral aneurysm (syphilitic). Abdomen opened and the common iliac controlled by digital compression. Obliterative operation. Patient examined in November, 1923, and there was no evidence of recurrence, and he had no pain in leg.

PRELIMINARY REPORT OF A LIGATION OF THE ABDOMINAL AORTA ABOVE THE BIFURCATION FOR AN ACUTE, RUPTURED, SYPHILITIC ANEURYSM INVOLVING THE ORIGIN OF BOTH COMMON ILIACS. (SURVIVAL OF THE PATIENT ONE YEAR, FIVE MONTHS AND NINE DAYS AFTER THE LIGATION.)

Death Caused by a Fulminating Pulmonary Hemorrhage from a Tubercular Cavity Developed in the Course of an Acute Generalized Tuberculosis. The Aortic Aneurysm almost Completely Obliterated by Dense Clot. The Process of Cure had been Practically Effected at the Time of the Fatal Pulmonary Hemorrhage.

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Synopsis of the Clinical Report. A bilateral aneurysm rapidly developed in both common iliac arteries, including the bifurcation of the abdominal aorta, in a young syphilitic negress, aged twenty-eight years. The bisaccular tumor projected from the promontory of the sacrum, pulsating vigorously in the hypogastrium up to the umbilical level and filling the true pelvis. Ligation of the abdominal aorta (April 9, 1923) immediately above the sac, with *completely* occluding half-inch cotton-tape ligatures in juxtaposition (one above the other). Immediate total arrest of all pulsation and circulation in the sac and of the femoral and pedal pulses in both lower limbs. Return of pulsation and aneurysmal bruit in the sac, with coincident pulsation in the femoral arteries at the groin, on the ninth day. In the course of a year a gradual

but progressive reduction in the size of the sac, with cessation of pulsation and of the aneurysmal bruit, except a limited area in the umbilical zone, followed by progressive hardening of the right (larger) sac and almost total obliteration of the right femoral pulse; the left sac still pulsating vigorously, but much harder and reduced in size. Left femoral pulse still felt, but beating feebly at the groin. No pedal pulses on either side. General improvement in the patient's condition under the influence of rest and specific medication, in spite of a series of complications which, at various times, threatened the life of the patient (bilateral lobular pneumonia; albuminuria; suppurative osteoperiostitis of the left hip—the greater trochanteric region—requiring incision and drainage of a large abscess; compression neuritis of the anterior crural nerve and branches, with consecutive contracture—flexion—of right leg). No sloughs, no decubitus eschars or trophic ulcers visible anywhere, the left lower limb preserving its nutrition, normal appearance and function, without disability in sensation or locomotion. Only a stiff and contractured right leg, with hyperesthesia and hard edema of the foot, remain to testify to a neuroparalytic and ischemic process which had been initiated in this extremity before the ligation of the aorta, the patient gaining in weight and strength, one year and eight days after the ligation of the abdominal aorta.

Summary of the Subsequent History of the Patient, following Preliminary Report Submitted to the Association at the Meeting in Baltimore, April 18, 1924. As stated in the preceding synopsis, the patient was gaining weight and strength and was able to walk about on crutches or to roll about the hospital in an invalid chair up to the date of the report, April 18, 1924.

In this report the clinical history and detailed description of the operation were supplemented by the postoperative record, illustrated by photographs of the patient, drawings, radiographs and charts, covering the whole period of twelve months and eight days following the ligation of the abdominal aorta. In the

author's commentaries and discussions of the postoperative history the theories that would account for the early return of pulsation in the aneurysmal sac and in the femoral vessels were especially considered. The pulsation in the sac had returned on the ninth day after the ligation, and with this return of pulsation and reappearance of the femoral pulses the rapid pulse and respiration that had characterized the first two weeks after the ligation gradually subsided from a pulse-rate of 160 on the first day to 96 on the twenty-first day. The general blood-pressure, taken in the arm, fell with the ligation from 118/90 to 100/75. The blood-pressure shortly after the ligation was 114/72, but gradually fell in the next few days to 80/60 and continued low for months during the whole summer. It was not until November, 1923, when the patient's strength returned and the heart shadows, as shown by the radiographs, had enlarged, that the blood-pressure rose to 112/70 and continued stabilized at about this rate up to the time of the report to the Association, in April, 1924.

The embarrassment of the pulmonary circulation following the immediate rise in the aortic tension after the ligation is also shown by the increase in the respiratory rate from 26 to 40 and 50 until April 27, when the respirations dropped gradually to 26. It is really surprising that the myocardium, enfeebled by a protracted and most virulent syphilitic infection, a generalized polyarthritis of the spine, hip and knee-joint, accompanied by a constant high fever for months, should have survived the continued strain of the aortic and pulmonary obstruction imposed upon the heart by the total occlusion of the abdominal aorta.

While the cardiopulmonary disturbances noted in this case can be easily accounted for by the strain upon the heart, and the improvement which gradually followed the reëstablishment of the aneurysmal circulation, with return of pulsation and bruit in the sac as early as the ninth day, is also explained by the relief in the aortic tension, the fact remains that the peripheral circulation in the extremities was well maintained in spite of the absence of all the distal pulses in the lower extremities during

the nine days that the aneurysm was pulseless and completely stilled.

But why did the pulsation return in the aneurysm on the ninth day after the unquestionably total occlusion of the aorta? That is the question.

In this case the aorta was ligated immediately above the sac on the proximal (cardiac) side of the aneurysm, the two tape ligatures actually encroaching upon the dilated cone formed by the artery as it merged into the aneurysmal sac.

The impending rupture of the secondary sac which had formed in the retroperitoneum as the consequence of a large leak which had occurred sometime previously, made it necessary to tie the aorta completely, not partially. That this was done effectively was shown by the collapse of the sac and the complete arrest of the femoral and of all the peripheral pulses. However, in spite of this, the lower limbs, to the toes, retained their warmth throughout and never showed the waxy, cadaveric pallor of total anemia. Evidently blood found its way to the extremities through collateral channels, even immediately after the aortic stream had been blocked by the ligature.

To account for the relapse in the aneurysm three theories were considered:

First, that, in spite of the double ligature and the care taken to prevent the knots from slipping by transfixing them with through-and-through silk sutures, it was possible that the texture of the material (half-inch cotton-tape for both ligatures, had stretched under the constant pounding of the systolic wave and the softening effect of the tissue juices, without the yielding of the knots. If this had occurred the early return of pulsation would be easily explained by the reëstablishment of the aortic channel sufficiently to allow a small, reduced stream to force its way from the aorta into the sac. While this would seem scarcely probable with tape bands tightened with sufficient force to crush the arterial walls, it was none the less conceivable that the fabric made of cotton fibre could have stretched sufficiently to allow a small stream to go through.

The second theory was that the aortic wall had been cut through in some part of its circumference, thus permitting the formation of an intra- or extramural passage between the newly formed capsule of organized exudate and the ligatures, thus restoring the continuity of the aortic blood stream through a newly created passage. This was not probable, as a fatal hemorrhage or pulsating hematoma would seem unavoidable.

The third explanation that suggested itself was that the collateral circulation, which had developed in consequence of the obstacle created by a large leaking aneurysm, was not only able to reëstablish the peripheral circulation, but to find its way back into the sac by retrograde paths—the large anastomotic branches of the internal and external iliac, lumbar, iliolumbar, with the circumflex iliac, sacral, gluteal, inferior hemorrhoidal, deep epigastric, etc.

Of these explanations, the first and third seemed the most plausible in accounting for the return of the pulsation in the sac and in the extremities. As the subsequent history has shown, the first of these theories proved to be correct, with the third as a contributing element, especially in accounting for the preservation of the circulation in the lower limbs during the first nine days after the ligation, when all the aneurysmal signs and the peripheral pulses were suppressed.

* * * *

History Subsequent to April 18, 1924. On my return to New Orleans from the meeting on April 20, I found that the patient had developed a slight evening fever, which was first accounted for by the presence of a group of enlarged lymph nodes in the left submaxillary triangle. At first we were inclined to attribute these to an oral infection originating in a carious tooth on the corresponding side. On April 28 the glands were removed by a block dissection, which completely emptied the submaxillary triangle. Coincidently the carious teeth were extracted. The gross and microscopic examination of the gland tissue showed unmistakable foci of caseous degeneration and

typical tuberculous infiltration. A careful examination was made of the abdomen on this occasion and the following observations were recorded on the operative sheet: "The original aneurysmal tumor for which the aorta had been ligated on April 11, 1923, has contracted enormously, diminishing fully 60 per cent of its original size, and the pulsations have disappeared over a corresponding area. A spherical mass, however, still pulsates on the brim of the pelvis, apparently a little below the promontory of the sacrum and over the left sacroiliac joint. This mass pulsates, but much less vigorously than after the beginning of the relapse. Its anatomical position suggests that it is a relic of the aneurysm corresponding to the left common iliac. The general expansile pulsation has certainly diminished very perceptibly, even in this circumscribed area. The patient has been practically free from the subjective symptoms caused by the aneurysm in the last two months. No pains in the pelvis or radiating pains down the lower limbs. Apparently the tumor is undergoing progressive obliteration by gradual deposition of clot."

After the submaxillary adenectomy the patient improved as the temperature diminished; but in June the fever returned, and with it another chain of glands appeared in the left cervical region, which spread rapidly along the superior and inferior cervical triangles. On July 1, 1924, under local and regional anesthesia (novocain-adrenalin), a long chain of densely infiltrated tissue glands was removed in block by a very extensive dissection. All the glands showed a very advanced stage of tubercular degeneration. The extent of the disease, following as it did the sheath of the carotid and the internal jugular into the anterior mediastinum, and the rapidity with which the infiltration had spread, made us fear that the deeper mediastinal groups were also involved.

Following the extensive adenectomy the patient developed a high, continued, remittent fever which could not be explained by the condition of the wound. This was reopened for inspection, with negative results as far as any infection was apparent.

The fever continued with remissions between 101° and 104° F., with cough and expectoration and all the signs of bilateral pulmonary consolidation. On July 31 the sputum showed tubercle bacilli, and subsequent examinations confirmed the diagnosis of acute pulmonary tuberculosis, with rapid breakdown of the lungs and formation of cavities. From this on the patient grew weaker, losing weight steadily. The blood-pressure fell progressively. Toward the end of July the fever abated, but continued with evening rises to 101° and 102°, followed by profuse sweats. The pulmonary signs of softening with cavity formation, cough and mucopurulent expectoration increased with extraordinary rapidity. On August 6 the patient's weight had dropped to 99½ pounds; blood-pressure, 90/60.

In spite of all this she insisted upon sitting up in a rolling chair. On September 3 she was transferred to the Tuberculosis Pavilion of the hospital. There she remained, constantly in bed, though cheerful and hopeful, getting thinner and weaker every day.

On Monday, September 10, 1924, at about 10 A.M., she was seized with a violent cough, which was accompanied by a great gush of blood from the lungs. She died in a few minutes, before the ward nurses or attendants could scarcely reach her side.

* * * *

Preservation and Roentgenologic Examination of the Cadaver before Autopsy. Immediately after death, at 10.30 A.M., September 10, 1924, the body was transferred to the morgue of the hospital and kept in cold storage for eight hours. Marked cadaveric rigidity had occurred. The arterial system was then injected with commercial (undertaker's) embalming fluid, consisting essentially of a dilution of formalin in alcohol.

The fluid had been tested and compared with other preservative fluids to determine its relative translucency to the *x*-rays. Three standard solutions used in the laboratory of the medical school of Tulane University were examined radiologically and all were found translucent to the rays, but the fluid selected was

particularly clear and shadowless in the radiographs of the three test-tubes subjected to examination in the *x*-ray laboratory of the hospital. The embalming fluid was injected through the brachial arteries, and it was noted that it reached every part of the body, proving that there was no serious impediment to the circulation even in the extreme periphery of the lower extremities, including the feet and toes.

The next day, September 12, the arterial system was again injected—this time with an opaque aqueous dilution of bismuth magma, which was allowed to flow by gravity into the ascending aorta, care being taken not to force the injection for fear of rupturing the aneurysm and causing extravasations that would obscure the view of the great arteries and of their collateral branches, which it was our purpose to study in the radiographs before attempting their anatomical dissection. With the invaluable coöperation of Dr. Granger and the staff of the *X*-ray Laboratory of the Charity Hospital, a series of radiographs (including the head, neck, thorax, abdomen and the extremities) was then made. The results obtained by these radiographs were very instructive and remarkable in many ways. They showed clearly: (1) That the main arteries of the lower extremities (iliacs, femorals, tibials) were pervious to their terminations; (2) that the opaque fluid entered the aneurysmal sac *directly* through the aorta, and that this found its way to the lower extremities by way of the common, external and internal iliacs, but chiefly through the left common iliac and its branches—the right common iliac having been obliterated in the sac; (3) the radiographs showed that the opaque fluid injected through the aorta had penetrated all parts of the arterial system, and proved that by this method a view of the arterial circulation could be obtained which was far more detailed than by an actual anatomical dissection.

Unfortunately the charting and tracing of the collaterals of the deep aortic branches were obscured by the overlying shadows of the visceral vessels, which were injected in great profusion, forming dense plexuses of minute and nameless arteries. This

was particularly noticeable in the lungs, coronary circle of the heart, abdominal and pelvic viscera and in the brain. This obscuring effect could have been avoided by the removal of the viscera after the opaque injection, and by other modifications of the technic which were not thought of or appreciated at the time. The crude results obtained by the imperfect application of the roentgenologic method in this case—probably the first in which it has been applied long after the surgical ligation of the aorta in the living human subject—fully confirms the experimental studies of Katzenstein (1905), Offergeld (1907), Mangin (1910), Bolognesi (1919), Orrin (1920), and more especially of Leriche and his associates (1922), and attests the immense value of the radiologic method in the study of the collateral circulation after the occlusion of the great surgical arteries.

Autopsy and Conclusions. On September 13, the third day after death, a systematis autopsy was performed, which was followed by a special dissection of the aorta and its branches, and of the aneurysmal sac in all its anatomical relations. Photographs of all the involved parts were taken *seriatim* as they were clearly exposed. The conclusions arrived at were:

1. That death had been caused by pulmonary hemorrhage. The bleeding had followed the erosion of a large vessel in the walls of a tuberculous cavity in the middle and lower lobe of the left lung which communicated with a large bronchus. Both lungs were dense infiltrated with tubercular deposits, which completely consolidated the whole left lung and the base and posterior half of the right lung. The tubercular mass in the left lung had broken down and formed a series of cavities which honeycombed the center of the left lung where the hemorrhage had occurred. The pleural cavities were obliterated by dense adhesions.

2. The posterior mediastinal lymph nodes, those along the thoracic duct from the neck to the diaphragm, and the retroperitoneal and mesenteric nodes were infiltrated and enlarged by recent tubercular deposits, which testified to the acuteness and wide range of the infection.

3. The *aneurysmal sac* formed a prominent, hard mass, which bulged as large as an adult fist in the midline overlapping the bifurcation of the aorta. It was evident that the sac had begun to leak a long time before the ligation of the abdominal aorta. The extravasations had formed a number of large diverticula, which extended laterally far into the right and less into the left iliac fossæ, and downward into the hollow of the sacrum as low as the third sacral vertebra. The body of the last lumbar vertebra and anterior surface of the sacrum had been eroded and denuded of periosteum, but were now covered with a layer of firm, laminated clot. The right iliopsoas muscle had been completely disintegrated and the sac wall was formed by the iliac fascia. The primitive sac had formed at the exact level of the bifurcation involving the origin of both common iliacs. It had ruptured at a very early stage of its formation, the original sac, which was well defined, probably not attaining the size of a small hen-egg when it began to leak. Therefore the bulk of the large sac recognized in life and revealed in the cadaver was formed by a number of secondary sacs or diverticula which encapsulated the extravasations as they spread in lines of least resistance under the iliac fascia and the retroperitoneum of the sacral hollow. The secondary sacs were filled with dense clot, which was laminated for the greater part and was so densely adherent to the bone and sac walls that it had to be scrubbed vigorously to detach it. It had evidently formed and hardened before it had felt the coagulating and hardening effect of the formalinized embalming fluid. The clot weighed, when removed from the sac, a little over 300 gm.

4. On investigating the relations of the aorta to the sac at the seat of the ligation, this was recognized only by a spindle of dense fibrous tissue which completely enveloped the aorta immediately above the fusion of the artery with the sac. The two half-inch woven cotton tapes which had been used to ligate the aorta were concealed from view by the encapsulating mass of fibrous tissue and organized exudates in which they were imbedded. They had not ruptured the intima and were not

in the least visible in the lumen of the aorta. The only indication or sign of their presence was a circular elevation or diaphragm formed by the inner wall of the artery where the aorta had been occluded. In the center of this diaphragm, which was smooth and perfectly covered by the endothelium of the intima, and free from clot, there was a small, narrow, central opening, invisible from the cardiac side, which allowed the tip of a uterine probe to pass through the stricture with some friction. This opening was about 8 mm. in circumference (estimated by the calibre of the probe), and it was through this small opening that a stream of blood had found its way directly into the aneurysmal sac with sufficient force and volume to feed the sac and to nourish the peripheral parts.

On completely dividing the aortic walls through this diaphragm, the two tape ligatures were found at the level of the constriction. They had diminished considerably in bulk, but were perfect in their continuity. The knots, which had been transfixed with silk sutures to prevent slipping, had not yielded in any way. It was evident, however, that the tapes had relaxed as they softened under the combined effect of the tissue juices and the infiltration of the fabric with invading tissue cells. In this way a total occlusion was gradually transformed into a narrow stricture, thus accounting for the return of the aneurysmal pulsation and the relief of the cardiopulmonary strain, which had caused so much anxiety during the first nine days following the ligation.

The fact that the circulation in the lower extremities had been well maintained during the period of total occlusion (nine days) shows that the collateral circulation had developed sufficiently before the ligation to insure the vitality of the lower limbs, though not enough to prevent an undue and almost fatal strain upon the heart.

5. The great improvement in all subjective symptoms and in the objective signs of the aneurysm (progressive reduction in the size of the aneurysm, increasing hardness, contraction of the pulsating area, diminution in the extent and intensity of the

aneurysmal bruits) which were most noticeable toward the end of the patient's life, as the blood-pressure fell with the advancing tuberculous infection, strongly suggests that a complete consolidation and cure of the aneurysm were about to be effected when the patient's life was cut short by the overwhelming tubercular infection—fulminating pulmonary hemorrhage—one year, five months and nine days after the ligation.

The clinical evidence to this effect was fully confirmed by the postmortem findings, which showed that the sac had filled with firm, laminated clot and that the aortic blood stream reached the lower extremities only through an open channel in the sac that led to the left common iliac—the right common iliac having been obliterated in the walls of the sac.

Conclusion. This observation shows that even though a primary total (atresic) occlusion of the abdominal aorta, through the relaxation of the ligature material, gradually becomes a *partial* (stenotic) occlusion, the much reduced circulation in the sac which follows this relaxation is not necessarily prohibitive of cure, but may in reality prove of advantage in favoring a more gradual but firmer consolidation of the clot. Furthermore, the gradual yielding of the ligature material in weak, exhausted patients with feeble hearts, ill-prepared to stand the enormous strain of a total aortic occlusion, as was notably the case in this patient, may save a heart that would otherwise have failed if a more unyielding material had been used for the occlusion. Again, the facts revealed by the anatomical and histologic study of the aorta at the seat of the ligature conclusively prove that tape ligatures made of cotton fabric, when applied with sufficient force to completely occlude the lumen of the aorta, remain encysted in the walls of the artery and are perfectly tolerated by the tissues. In this case the tapes remained imbedded in the aortic walls as a constricting ring for over seventeen months without causing the slightest necrotic or degenerative changes in the intima; in fact, the vessel walls were strengthened by the dense capsule of organized exudates and fibrous tissue of new formation in which the tapes were imbedded. In view of the

perfect tolerance of this material by the bloodvessel wall it would seem superfluous to resort to strips of fascia or aorta or other less readily available or dependable (shorter-lived) material for the purpose of occluding a large artery of the calibre of the aorta. On the other hand, ligatures made of more rigid or unyielding materials (metallic bands), which will maintain a complete atresic occlusion of the aortic lumen, are more likely to cause premature ulceration, atrophic or necrotic changes in the arterial wall which may end in disastrous hemorrhages. For this reason the writer still holds to cotton or silk tape as the safest and most practical material for the ligation of the aorta and its great primary branches (innominate, left carotid, left subclavian, common iliacs, at their origin), as first taught by the late Professor Halsted in the latter part of his career.

A final report of this case in all its bearings, including the radiographs, photographs, photomicrographs and drawings, showing histology of the aorta at the seat of ligation, is in course of preparation and will appear in a later publication.

ANEURYSM OF THE INTERNAL MAMMARY ARTERY

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(By Invitation)

The internal mammary artery coming off from the subclavian vessel opposite the thyroid axis pursues an extrapleural course through the thorax to about the junction of the sixth rib with its costal cartilage, where it divides into two trunks, the musculophrenic and the superior epigastric, the latter of which completes the anastomosis between the upper and lower sections of the body by joining with the ascending branch of the deep epigastric. The anatomical protection of the chest wall renders this vessel less liable to injury than the more superficial vessels of the extremities, and yet occasionally it is divided by a gunshot or stab wound, which in addition almost invariably injures some of the thoracic viscera. Following such an injury a false aneurysm or pulsating hematoma is rarely seen. Weiting notes 2 cases of false aneurysm of this vessel seen in chest injuries during the recent war. True aneurysmal formation, however, is an exceptionally rare occurrence, as a search of the literature reveals only 3 cases of this nature. In each instance there was a failure of diagnosis, 1 case being mistaken for an aneurysm of the innominate artery, or arch of the aorta, by one physician, but diagnosed and operated upon for an abscess of the chest wall by another. The other 2 cases were also operated upon with a mistaken diagnosis, and all 3 terminated fatally. The case which stimulated my interest in this condition came to the dispensary January 24, 1923, with a pulsating tumor the size of a hickory-nut, which presented in the third interspace about

one inch from the sternal border. A diagnosis of aneurysm of the right internal mammary was made after considerable consultation and the lesion treated by extirpation of the aneurysmal sac, which was followed by a prompt and rapid recovery. The case report is as follows:

D. O., colored, female, aged forty-one years, married.

Chief Complaint. Swelling on right side of chest; some sense of pain around heart.

Family History. Father dead from tuberculosis, aged fifty years. Mother dead from tuberculosis, aged forty-nine years. Two brothers living and well; one dead from meningitis. Two sisters living and well; two sisters dead from tuberculosis. Two children living and well. Has had measles, mumps, whooping-cough.

Past History. Patient has been in good health until July, 1923. At sixteen years had typhoid fever, with complete recovery. At twenty-one years she had smallpox. Has dull headaches occasionally, which last for about two days. Patient complains that she gets blind when she stoops at times, and notes that headaches precede her menses. There is no alopecia.

Eyes: Some disturbance of vision; has to wear glasses.

Ears: Negative.

Nose: Negative.

Mouth: Has recently had all of her teeth extracted.

Neck: Negative for thyroid enlargement. There is no tracheal tug. The glands in the posterior triangle of the neck are palpable, but small.

Cardiorespiration: Has some cough at times over four or five years; no dyspnea. Patient thinks she has spit up some blood and had some night-sweats, but physical findings and *x*-ray do not reveal evidences of pulmonary tuberculosis. She has lost 30 pounds in weight since last July.

Gastrointestinal: Negative. Patient has been constipated for some years.

Genito-urinary: Negative, except for some frequency of urination.

Venereal: Patient had a yellowish vaginal discharge a few years ago, but this cleared up under treatment. She denies syphilis or other venereal disease.

Menstrual History. Menses began at sixteen, but prior to that time she had bled vicariously through the nose several times. Up to three years ago her menstruation was regular, occurring once a month and lasting three or four days. For the past three or four years her menses have been very irregular, occuring twice a month and lasting seven or eight days. Some leucorrhea, which is odorless.

Marital History. Has been married twenty-two years. Has had seven pregnancies; all labors difficult, usually lasting twenty-four hours or more. She has two healthy children, twenty-one and eighteen years of age, respectively, and has had five miscarriages.

Present Illness. In July of the present year patient noticed a small pulsating tumor along the right side of the sternum. This tumor began to increase and has progressively and slowly enlarged since. About one month ago she noticed dull pain and tenderness in the entire upper portion of her chest anteriorly. She has had some vertigo occasionally. For the past week has had some dull, aching pains in the precordial region. They seem to begin in the axillary line and advance to the sternum, being more marked under the left breast. She has lost thirty pounds in weight in the past seven years, her best weight being 140 pounds; her present weight is 110.

Physical Examination. The examination of the tumor mass shows a pulsating expansile tumor of the right anterior thoracic wall in the third interspace over which a distinct bruit and thrill can be made out. The tumor is about the size of a hickory-nut and may be picked up between the fingers, apparently having no attachment to the chest wall. There is no difference in the radial pulse on either side; pulsation in the tumor is synchronous with the radial heat. The blood-pressure on each side is 115/80. The skin over the tumor is movable and there is no evidence of inflammation.

Fluoroscopic Examination. Shows a normal heart and aorta and shows nothing in the area occupied by the tumor. Wassermann, is positive, four plus.

Urinalsysis. Specific gravity, 1020. Negative for albumin and sugar. Microscopic examination shows few pus cells and numerous epithelial cells. No casts.

Blood. Hemoglobin, 85 per cent. White blood cells, 7500.

Operation. Under ether anesthesia an extirpation of the tumor mass was made on January 24, 1923. A longitudinal incision parallel to and about one inch from the outer border of the sternum, and extending from just below the clavicle to a point about one inch above and external to the ensiform, gave ample working exposure. The first step in the operation was to expose the mammary vessels in the first interspace in order to control any bleeding that might occur. These vessels, both artery and vein, were found to be dilated, the artery being about the size of a normal superior thyroid and the veins correspondingly enlarged. By pressure over the vessels all pulsation in the tumor was stopped. Owing to the rich collateral circulation it was not felt that simple ligation would suffice. I tied the vessels here with catgut and then removed portions of the second, third, fourth and fifth ribs for about one inch back of their junction with their costal cartilages and elevated this flap until it was possible to tie the superficial epigastric below the tumor. The corresponding intercostal vessels were ligated as they were approached. By blunt dissection I was able to free the pleura from under the sternum and to get a finger around the entire tumor mass. The costal cartilages were then excised close to the sternum and the aneurysmal sac with the portion of the chest to be sacrified was removed as one specimen. The sac was found to hold about one ounce of fluid and to emerge between the costal cartilage of the third and fourth ribs. There was no bone necrosis at the costochondral junction. The wound was closed without drainage.

The patient made an uninterrupted convalescence, and was dismissed from the hospital on the tenth day.

Discussion. The first case that I have been able to find in the literature was that of Sickles, who reported, in 1847, a case which he had seen eight years previously. This case presented a small tumor of the left chest wall which emerged between the third and fourth ribs, and which was about the size of a common marble. By pressing the finger hard upon it he was able to make it disappear into the chest. From its expansile pulsation and the bruit a diagnosis of aneurysm was made, but he felt that the aneurysmal formation was in the aorta or innominate vessel. The patient subsequently went to another physician and a diagnosis of abscess was made and the tumor was lanced. The ensuing hemorrhage was controlled by pressure, but recurred eleven days later with a fatal termination. The autopsy proved the case to be an aneurysm of the internal mammary vessel and not of the aorta. The autopsy also revealed that there was some absorption of the fourth rib and a portion of the sternum.

In 1896 Campos Hugueney reported a case of a man, aged fifty-four years, who presented himself with a tumor of the right chest wall about the size of a walnut, about opposite the sternum in the third interspace. This had been diagnosed a sebaceous cyst, and was incised. The hemorrhage which had occurred was controlled by direct compression and tampons of hyperchloride of iron. Eleven days following this, when seen by Hugueney, the tumor presented a large ulcerating mass with gangrenous odor, without bruit or expansion; pulsations were synchronous with the radial, and the consistency of the tumor was that of a sac and fluctuant. Recognizing the condition, he ligated the internal mammary vessel in the first interspace and abandoned extirpation of the tumor mass. The patient died sixteen days later of a recurrent hemorrhage. Autopsy revealed a true aneurysm of the internal mammary vessel. The case of Reynes, published in 1909, was very similar to the two mentioned before. The difficulty of diagnosis is apparent from the notes he makes on the case, and despite considerable consultation, he was unable to decide definitely between aneurysm of the aorta, a cold abscess or a pulsating empyema. Finally, aneurysm

was excluded and the tumor mass opened, followed by a severe hemorrhage. Again this was controlled by pressure, but recurred three days later, and the patient died.

FIG. 1.—Photograph showing internal mammary aneurysm projecting in third right interspace.

The aneurysmal sac was typical of this type of case, the vessel having made a small opening into the tissues and then formed

a false aneurysm which extended along the ribs communicating with the artery by a narrow neck. On the interior of the thorax was a true aneurysmal sac which he aptly describes as an aneurysm like a button sewed on a shirt. The artery below the aneurysm was obliterated.

It is evident from the observations of the men reporting these cases that a diagnosis is not always apparent, even after careful consideration, and because the roentgenological findings are not

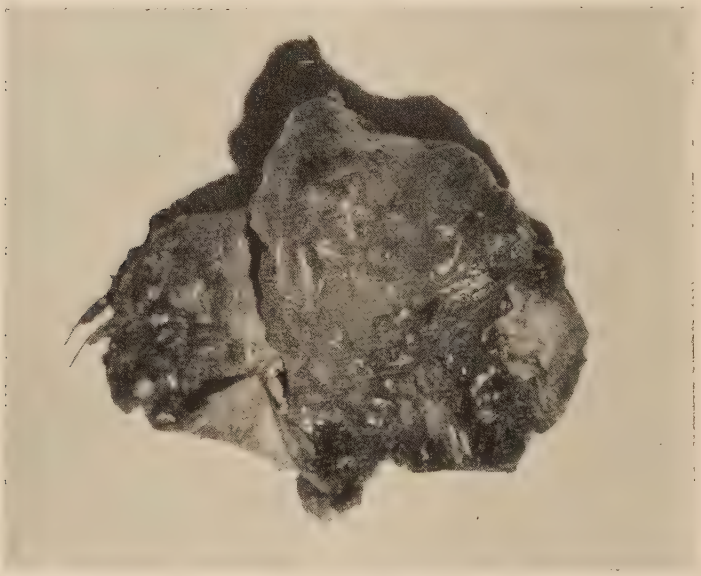

FIG. 2.—Gross specimen, anterior view showing the sac on the external chest wall, with the resected ribs and costal cartilage.

conclusive one is frequently confused by several possibilities. Obviously an expansile tumor in any portion of the body over the course of any bloodvessel should be suspected of being an aneurysm. In this particular location, where the most frequent aneurysm is that of the arch of the aorta, the *x*-ray is of value in determining the size and position of the heart and great vessels of the thorax. In our case the fluoroscopic findings demonstrated that the heart was normal in size and position and the aorta

was normal; the area over the tumor mass showed no difference in density. A pulsating empyema or cold abscess over the ribs themselves would ordinarily be differentiated by the roentgenological findings. The sphygmograph should be a very material aid in deciding the diagnosis by giving the characteristic

FIG. 3.—Posterior view of same specimen with probes in the mouth of the vessel.

deformity of the radial pulse in the case of an aortic aneurysm; also the anatomical level of the tumor in the third interspace should be sufficient to reject the hypothesis of aortic aneurysm. Reynes points out a sign which he believes will enable one to make a diagnosis between an aortic aneurysm and an aneurysm

of the internal mammary. I quote his article: "Aneurysm of the internal mammary is of such rarity that one should stop and argue the possibilities of such a thing. If there is aneurysm, one would be more liable to think of the aorta. However, there are two signs against this diagnosis: First, aortic aneurysm; the location of the tumor in the third interspace instead of the second, which indicates at least that the aneurysm is very small. Second, the propagation of the systolic bruit is very harsh, especially at the level of the tumor. This bruit follows very nearly the direction of the third and fourth ribs in the axilla and absolutely does not ascend toward the clavicle." He believes that the direction of the bruit toward the axilla can be considered a good differential sign between aortic and internal mammary aneurysm.

The diagnosis once established, the question of what surgical treatment to pursue arises. In one of these cases reports it is noted that the simple ligation of the internal mammary vessel in the first interspace was followed by a reëstablishment of the collateral circulation sufficient to cause a secondary hemorrhage, which resulted fatally. The rich anastomosis between the intercostals and phrenics below suggests the impossibility of cure without radical extirpation of the sac. The resection of a portion of the chest wall and the costal cartilage, following a preliminary ligation of the vessel in the first or second interspace, and also below the aneurysm, with a total extirpation of the sac, is a feasible procedure which may be carried out without injury to the pleura or danger of unpleasant sequelæ.

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REPORT OF A CASE OF AXILLARY ANEURYSM SUCCESSFULLY TREATED BY ENDO-ANEURYSMORRHAPHY (MATAS)

WITH AN ANALYSIS OF THE CASES OF ANEURYSM TREATED AT THE MASSACHUSETTS GENERAL HOSPITAL FOR THE PAST FIFTY YEARS

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After a recent case of axillary aneurysm successfully treated by the Matas method, I became so thoroughly convinced of its simplicity and sound principles that I regret that more cases have not been similarly treated at the Massachusetts General Hospital, for I believe it to be the ideal method, always to be attempted, and abandoned only for definite contraindications. Dr. J. C. White has aided me in a brief analysis of the cases of aneurysm of the larger arteries distal to the aorta which have been surgically treated at our Hospital during the past fifty years. I have considered, particularly in the early cases, compression and tourniquet as surgical treatment. The total number is much smaller than I had imagined, only 56 cases.

In the first period, from 1872 to 1884, there were 3 cases of subclavian aneurysm treated by compression or tourniquet. Two were improved, but not cured, and a third developed paralysis of the arm and died on the second day. One case of brachial aneurysm was successfully treated by Henry J. Bigelow, in 1872, by proximal and distal ligation. There were 6 cases of femoral aneurysm, all cured, 1 by tourniquet for twenty-four hours and 5 by distal and proximal ligation, which I shall speak of hereafter

as double ligation. Five were popliteal aneurysms, all treated by tourniquet; 4 discharged without pulsation; 1 died on the following day.

In the second ten-year period, from 1884 to 1894, there was 1 case of subclavian aneurysm with ligature of the axillary and carotid, which died from rupture on the eighth day. One case, in 1891, was a common iliac aneurysm, distal and proximal ligation, with death in six hours. There were 3 cases of femoral aneurysm, all well, 2 recovering after distal and double proximal ligation; 1 after ligation of the external iliac. There were 6 cases of popliteal aneurysms: One died from sepsis a month after ligation in Hunter's canal; 5 were cured: 1 after tourniquet; 2 by distal and double proximal ligation; 1 by ligation in Scarpa's triangle; 1 by proximal ligation, followed, however, by gangrene of the leg, which necessitated amputation.

From 1894 to 1904 there were 2 cases of axillary aneurysm treated by ligature of the first portion of the subclavian: One with distinct improvement, but no cure, and the other, followed by gangrene of the forearm; after amputation at the elbow the patient recovered. One case of brachial arteriovenous aneurysm was treated by double ligation and excision of tumor and cured by Dr. Mixter. There were 2 cases of femoral aneurysm cured: One by proximal and the other by proximal and double distal ligation. There were, again, 7 cases of popliteal aneurysm: One died of shock after distal and double proximal ligation; 1 was cured by ligation in Hunter's canal; 3 by distal and double proximal ligation of the artery and vein; 1 after distal and double proximal ligation, with excision of the sac, developed gangrene three days later, and the thigh was amputated. Another with double distal and proximal ligation and persistent pulsation from a collateral branch demanded immediate amputation of the thigh.

In the period from 1904 to 1914 there were only 2 popliteal aneurysms: One cured by double ligation and the other treated by immediate thigh amputation. There were 3 cases of femoral aneurysm: One cured by double ligation of the artery and vein,

another relieved by proximal ligature of the artery and vein, and the third operated upon as an emergency for a ruptured aneurysm in mid-thigh, died on the table. On July 27, 1912, I operated upon a left common carotid aneurysm by ligation of the artery with silk below the sac. On the following day there was still pulsation in the tumor, which slowly increased until August 15, when I performed ligature of the common carotid above the aneurysm and just below the birfurcation. Pulsation ceased, there was slight cyanosis. On the following day, however, the pulsation returned, though in less degree. In two days there was some aphasia and right hemiparesis. This had improved at the end of a week, but on September 7, three weeks after the second operation, the tumor was obviously increasing in size with more pain. Accordingly, under novocaine, I inserted ten feet of German silver wire into the sac with electrolysis (20 ma. for twenty minutes and 25 ma. for forty minutes). Two days afterward, on September 9, as no improvement showed itself, it was decided to operate again. In endeavoring to free the lower pole of the tumor the sac ruptured and a gush of blood escaped from an aperture at its lower end. While the artery was controlled proximally by an assistant's finger, an attempt was made to obliterate this opening within the sac with a curved needle and silk, but the walls were *too friable* for suture. The dissection was therefore continued downward along the common carotid, and finally a silk ligature was placed about the artery which completely controlled the hemorrhage. The patient, however, did not rally, and died on the following day.

There was 1 case of external iliac aneurysm operated upon by Dr. Balch and Dr. Frederick Murphy, July 21, 1908, the first case of endoaneurysmorrhaphy, according to the Matas method performed at the hospital. In the right iliac fossa there was a rounded, tense, pulsating swelling, about 4 inches in diameter, filling the hollow of the right iliac bone and extending to Poupart's ligament. A Crile clamp was applied to the common iliac artery and the sac opened. Large masses of laminated clot were removed, disclosing the opening from the

external iliac artery, about 1 inch above Poupart's ligament. This aperture was closed with fine silk sutures drawn through vaseline. Upon removal of the clamp there was no bleeding. A flap from the anterior part of the sac was then sutured with catgut to the repaired artery wall and the cavity of the aneurysm obliterated by further suture; gauze drainage. Immediately after the operation the circulation of the leg improved and pulsation in the dorsalis pedis returned. With the exception of some discharge of blood-clot and sac wall, convalescence was excellent, and five months afterward the circulation below the aneurysm was normal.

Innominate Aneurysm. In the period 1914 to 1924 there were 2 cases of aneurysm of the innominate:

Carrie S., 1915, aged sixty-three years. A year ago the patient, when excited, noticed throbbing above the right clavicle. Three weeks ago, a pulsating tumor the size of a walnut; blood-pressure, 180; Wassermann, negative. Operation by Dr. Cobb; novocaine anesthesia. Fusiform aneurysm was found involving the distal part of the innominate and proximal carotid. The innominate artery was tied below the aneurysm with double No. 15 silk; the carotid artery was tied distally with single silk ligature. Within twenty minutes complaint of slight numbness and tingling in the right hand. Convalescence was normal until the sixth day, when transient paralysis of the left arm and leg occurred. On the following day complete left hemiplegia, with cerebral symptoms, and death on the twelfth day.

Second case, 1923, a colored woman, aged forty-two years, with tertiary syphilis, had complained of a pulsating tumor in the lower right neck for five years. Specific treatment for three months. Examination showed a rounded pulsating tumor above the inner end of the clavicle, 4 to 5 cm. in diameter. Operation by Dr. Lincoln Davis; novocaine anesthesia. The innominate artery was found to have risen high in the neck and was two or three times the normal size—a case of arteriectasis. The carotid

was long and bent on itself in an S-shaped manner. An aluminum band was placed about the carotid at its junction with the innominate and pressed together until pulsation ceased. The aneurysm was not affected. One year afterward the patient complained of no symptoms; pulsation still present but distinctly less.

Subclavian Arteriovenous Aneurysm. W. H. S., 1916. In 1915 the patient was shot through the right clavicle with a revolver; wound infected. At the end of a week comminuted fragments of bone were removed. Three months later more necrotic bone was removed. Patient entered the Massachusetts General Hospital, West Medical Service, February 21, 1916, for pain and swelling of the arm. There is a palpable thrill over the area of the right clavicle and below it, with a loud systolic and diastolic murmur, transmitted over the chest and precordia. Right pulse weaker than the left. He complains of sharp pain running down the arm to the ring and little finger, which is becoming more intense. His right hand becomes cold more easily than the left. The whole of the right arm was definitely swollen. Anesthesia in the ulnar distribution in the forearm and hand. On slight pricking of the skin there was immediate bleeding. The patient returned for operation on May 1 of the same year. The symptoms had increased and there was now no pulse at the wrist. I made a vertical incision over the center of the clavicle, the mid-third of which was removed with rongeur forceps and Gigli saw. The muscles were retracted, bringing the subclavian vein into sight and revealing a sac, about the size of a golf ball, under the pectoral muscles. This was carefully freed until it could be displaced upward. No good view was ever obtained of the subclavian artery, but it was felt pulsating beneath the vein. Rubber-tipped clamps were then placed on the artery and vein above and below the sac. Crile clamps were also placed on the cephalic vein and a more distal branch of the subclavian as it entered the main trunk just opposite the aneurysm. The sac was then opened, showing a pedicle about three-fourths of an inch in

diameter, through which, however, one could not see the communication between the artery and vein. This neck was closed off with interrupted vaselined sutures, the sac amputated and reinforced by further suture with chromic catgut No. 0. Upon removal of the clamps there was no bleeding. As the communication between the artery and vein still persisted, it was decided to ligate the artery above and below. The clamps were reapplied and an attempt was made to separate the artery from the vein. This, however, owing to previous infection and scar tissue, proved extremely difficult, and finally resulted in a bad tear at the junction of the internal jugular and subclavian veins, with sudden profuse hemorrhage. By finger and tampon this was finally controlled, but not until some air had entered the circulation. Under intravenous saline and ten ounces of blood the condition improved, but in repeated endeavors to tie off this lesion and remove some of the clamps, more blood was lost. Finally several sutures were passed about the bleeding vessels *en masse*, and with a few hemostatic forceps left *in situ* the bleeding was completely controlled. Collateral engorged veins made this operation extremely difficult. In spite of further transfusion the patient rapidly failed, and died within a few hours.

Brachial Arteriovenous Aneurysm. J. S., November 16, 1917, aged forty-two years. Twenty-two years ago the patient was stabbed in the left arm with a knife. Soon after she noticed a constant thrill. Overuse causes marked swelling of the whole arm, with pain, particularly on its inner side, and in the axillary region. The arm is definitely weak. Examination showed scar of old injury in the left upper arm, beneath which is a pulsating tumor the size of an English walnut. There is a marked thrill and double bruit, loudest at this point, which is transmitted upward into the neck and downward as far as the radial vessels at the wrist. Median basilic vein is dilated and pulsates. On November 21 I made an incision over the scar and found the artery much dilated and thick-walled, numerous

enlarged veins, the size of one's thumb, with many communicating cross branches also dilated and pulsating. The artery was tied with silk 1 inch above the sac, which was not opened. The thrill entirely ceased after the artery had been occluded. The patient was discharged twelve days after operation. Radial pulse very weak; no bruit; previously dilated veins are collapsed; relieved.

Right Femoral Aneurysm. T. McC., aged fifty-three years; police officer, Winchester, Mass., entered the Massachusetts General Hospital on September 10, 1923. Three months ago, while undergoing an examination, an induration was found in the right femoral region. This slowly increased in size, and for two months has been pulsating with intermittent, slight pain down the inner part of the thigh. Five days ago, with increase in the size of the tumor, pain became intense, requiring several doses of morphine in twenty-four hours. Six months ago a diagnosis, after a careful urological examination, was made of left renal tuberculosis and right tuberculous epidiymitis. The patient has lost 35 pounds. Wassermann is negative; hemoglobin, 80 per cent.

Physical Examination: In the right femoral region is a pulsating tumor, about 4 inches in diameter, with considerable surrounding edema. Pressure on the external iliac artery completely controls pulsation. There is slight edema of the leg and slight fulness of the superficial veins. The diagnosis of femoral aneurysm was obvious, but there was some doubt, in view of the negative Wassermann, as to its origin. It seemed possible that a tuberculous gland in the groin, secondary to the epididymis, might have eroded the posterior part of the artery. There had been no history of trauma; there was no arteriosclerosis.

On September 11, 1923, under novocaine, I made an incision above and parallel to Poupart's ligament and by bloodless dissection the external iliac artery was isolated and tied with No. 3 chromic gut. Pulsation below this point at once ceased. Further examination showed that the aneurysm had risen so high that

it would have been impossible to have tied below Poupart's ligament. Owing to the possible tuberculous origin, it seemed unwise to open the sac and attempt a Matas operation, and the question of tying the vein simultaneously was decided in the negative. There was no shock following the operation. The leg, which became cool, was surrounded with sheet wadding and hot-water bottles, which soon were replaced by electric light bulbs under a hood. By September 18, the leg was warm spontaneously, of fair color, with the veins on the dorsum of the foot prominent. On the 23d the wound was soundly healed, no pulsation in sac, which was somewhat flatter, but had shrunk very little in size. On the 26th, definite pulsation in the dorsalis pedis, followed by pulsation of the posterior tibial on the 28th. Discharged on October 2; aneurysmal mass definitely smaller; no pain. By November 1 the aneurysmal sac had entirely disappeared but the patient was suffering from uremia, from which he died on November 20.

Axillary Aneurysm (*Matas*). J. D. N., 1924, aged fifty-six years, entered the Massachusetts General Hospital February 23, 1924. Probably old lues, though Wassermann was negative. One year ago, after a backward wrench of the left arm the patient noticed marked ecchymosis of the axillary region, arm and forearm. There was rapid improvement and no symptoms until six months ago, when he first noticed a swelling in the left axilla, which slowly increased in size and began to beat, causing him, however, very little pain or disability. Blood-pressure, 120/90. In the left axilla is a pulsating tumor the size of a large grapefruit, with a systolic murmur and prominent veins over the left chest. The skin is slightly adherent over it, without redness or tenderness. Neither the brachial artery nor the radial pulse can be felt. On March 5, under ether, I made a curved incision along the anterior border of the axilla and down the arm. The pectoralis major tendon was divided one inch from its insertion. Without difficulty the axillary artery was isolated from its accom-

panying veins and nerves and closed with a Crile clamp. The brachial artery was similarly treated two inches below the tumor. The sac was then opened and two handsful of laminated clot, like placenta, removed. After the inner surface had been

FIG. 1.—J. D. N. Axillary aneurysm. Before operation.

wiped clean, slight oozing was seen to occur from both ends of a gutter 1 inch long by $\frac{1}{4}$ inch wide, communicating with the axillary artery. The lateral walls of this gutter were firm, and most easily sutured with paraffin silk on a small curved needle,

and reinforced by a second row of continuous silk. There was no bleeding upon removal of the clamps. Much of the sac was then excised and the remainder obliterated by mattress stitches of small chromic catgut. Some of the redundant skin was then

FIG. 2.—The same, showing incision.

excised and the wound closed with silk; a small rubber drain was inserted; gauze pad and a Bender bandage. In twenty-six days the patient was discharged well, with a small area of necrosis of the skin and a definite radial pulse.

In addition, there were 6 cases of popliteal aneurysm during this period. In 1915 I attempted to perform a Matas obliterative operation, but the tissues in the sac were *too friable* for suture.

FIG. 3.—J. D. N. Obliterative endoaneurysmorrhaphy, Matas' method. Three weeks after operation.

I therefore ligated the vessels above the aneurysm and closed the sac with catgut sutures. Upon removing the tourniquet there was much venous hemorrhage from the popliteal vein, which was therefore ligated. Gangrene of the foot developed and a Chopart

amputation was performed five weeks after the operation. This was not adequate and one month later amputation of the leg became necessary. In 1916 Dr. Cobb performed a perfectly successful Matas obliterative operation, with normal convalescence. In 1920 a patient presented a large pulsating tumor with much surrounding inflammatory exudate and marked swelling of the leg. Under local anesthesia I applied a single ligature close to the aneurysm. Gangrene slowly developed, requiring amputation of the leg three weeks later. In 1921 Dr. D. F. Jones found a small aneurysm adherent to the popliteal vein. The artery proximal to the sac pulsated very feebly. The aneurysm itself did not pulsate nor did the artery distal to it. Proximal ligation with silk. Discharged well on the tenth day. In 1922 Dr. Edward Richardson performed a typical and successful Matas operation, with normal convalescence.

Popliteal Aneurysms

2 deaths, 7 amputations, 17 cures (2 Matas) 26

Femoral

1 death, 1 relieved, 12 cured 14

Iliac

1 common iliac, 1 death 1
1 external iliac, 1 cured (Matas) 1

Brachial

1 relieved, 2 cured 3

Axillary

1 relieved, 1 amputation elbow, 1 cure (Matas) 3

Subclavian

3 deaths: 1 arteriovenous
1 after ligature of carotid and axillary
1 tourniquet
2 relieved by tourniquet 5

Innominate

1 death, ligation innominate and carotid, 1 relieved (band on carotid) 2
1 common carotid, death after fourth operation 1

56

9 deaths, 8 amputations, 6 relieved, 40 cured.

These are the results by a general staff during the long period of fifty years. If one examines the recent cases critically and

impersonally, only one conclusion seems to me justifiable, that endoaneurysmorrhaphy, as advocated by Dr. Matas, with all preliminary precautions—prophylactic hemostasis—the aluminum band when indicated, is always the method of choice.

The only exceptions may be cases in which the sac walls are too friable for suture and a small number of arteriovenous aneurysms in which quadruple ligature may be necessary.

DISEASES OF THE BLOODVESSELS OF THE EXTREMITIES: DESCRIPTION OF TWO METHODS OF EXAMINATION

By BARNEY BROOKS, M.D. (By Invitation)
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THERE is at the present time no accurate method of estimating the extent of diminution in the circulation of the extremities resulting from disease of the bloodvessels. Furthermore, it is often impossible, by methods of examination now used, to determine if actual circulatory disturbance exists. The absence of any of the clinical manifestations of circulatory disease, with the presence of a good pulse in all the large arteries and normal color of the skin, may be taken as conclusive evidence of normal circulation. The presence of gangrene or even a marked blanching of the skin of an extremity is undisputed evidence of a deficient circulation of blood. Between these two extremes, however, are found the greatest number of instances of disease of the blood-vascular apparatus, and in these instances it is impossible to estimate with accuracy the degree of diminution of circulation or often to determine if any really exists. For example, a man, aged fifty-five years, who comes complaining of pain in the legs, may show none of the gross manifestations of arterial disease, and it cannot be determined if the pain is the result of "bad posture" or "bad circulation." Also, if the grossest manifestation of deficient circulation—for example, gangrene—exists, the degree of disturbance of the circulation in the adjacent tissues is a matter of conjecture. This is of great importance in the determination of the advisability of amputation and the selection of its level.

From the clinical standpoint there are two important facts to be determined in the examination of an extremity affected by obstruction of the arteries: first, the volume-flow of blood through the tissues, and, second, the site and extent of arterial obstruction.

The purpose of this paper is to describe some recently developed methods for these determinations.

METHOD 1. The first method to be described is analogous to the well-known hyperemia test of Moschkowicz. In both tests a study is made of the effect of the application and removal of a tourniquet. In the Moschkowicz test the criterion is color change. In the test to be described in this communication the criterion is change in temperature. The apparatus used consists of a thermocouple mounted in a needle and a sensitive galvanometer. With this apparatus changes in temperature of the deeper tissues can be measured with great accuracy.

The extremity to be examined is exposed for a period of a half hour in a closed room. The thermocouple needle is inserted into the tissue to be examined. Galvanometer readings are made until the temperature has assumed a constant level. A tight tourniquet is then applied to the extremity. Immediately the temperature in the tissues distal to the tourniquet begins to fall. When the tourniquet has been in place for ten minutes there is a fall of usually 2° to 5° C. The tourniquet is then quickly released and the galvanometer readings recorded at one-minute intervals. In the individual with no obstruction of his arteries the release of the tourniquet is always followed by a rapid rise of the temperature to or above the level at which the tourniquet was applied. In the individual with obstruction of the arteries of the extremity the release of the tourniquet does not result in rapid rise of the temperature. There may elapse a period of as much as seven minutes before the temperature begins to rise, or in tissues severely deprived of arterial blood no rise at all may be observed. Any appreciable period of time elapsing between the removal of the tourniquet and the beginning of the rise in temperature of the distal tissues has always been associated with demonstrable obstruction of the large arteries of the extremity

examined. The length of this period and the rapidity with which the temperature rises after it has once begun is an index of the condition of the arterial blood supply of the tissue examined.

METHOD 2. The second method has already been described and I repeat it here only because it has very recently been published.

If the application of the first method described indicates the existence of arterial obstruction, the site and extent of this arterial obstruction can be determined by injection of sodium iodide into the arteries, and *x*-ray examination. This method has been used approximately twenty-five times and has as yet shown no manifestations of being harmful. I am as yet, however, extremely careful in the selection of cases in which this method is used and have been very cautious concerning the quality of the sodium iodide solution used and the time which it is permitted to remain in the arteries.

The intra-arterial injection is carried out as follows: If the arteries of the lower extremity are to be examined, the femoral artery is exposed with a local anesthetic and an *x*-ray plate is put under the leg. The *x*-ray tube is put in position to obtain an *x*-ray photograph of the popliteal artery and its branches. A tourniquet is tightened about the thigh proximal to the incision just enough to cause the veins to fill. The femoral artery is then occluded either by a clamp or between the fingers, and 10 cc of sodium iodide solution injected into the artery distal to the occlusion. Immediately following the injection the *x*-ray tube is operated. The artery clamp and tourniquet are removed. It is best to have the patient under the influence of nitrous oxide gas during the brief period in which the sodium iodide solution is in the arteries.

The clinical application of these methods is illustrated by the following lantern slides:

DISCUSSION.—From the application of these two methods we believe it is possible to determine the actual condition of the arterial circulation of an extremity more accurately than heretofore has been possible. The time at my disposal does not

permit me to discuss the possibilities of treatment of the various sorts of obstructing diseases of the arteries. It is my belief, however, that heretofore all surgical procedures intended to benefit individuals suffering from the various clinical manifestations of arterial obstruction have been, so to speak, of the "hit" and "miss" variety. For example, the now much practised operation of Leriche is usually done without any exact knowledge of the condition which it is intended to relieve. If an artery is obstructed there is no value in a dilatation of the artery beyond the site of the obstruction. The arteries proximal to the obstruction which happen to be supplying the collateral circulation are the arteries which an attempt should be made to dilate. It therefore follows, if there is anything to periarterial sympathectomy, beneficial results could only be obtained by knowing where the operation should be performed. Furthermore, it is my belief that there is too much vague thinking about vasomotor phenomena and too little knowledge of the anatomical-pathological condition of the arteries.

HYDRAULIC PRINCIPLES AS APPLIED TO ARTERIO-VENOUS ANEURYSM: THEIR IMPORTANCE IN THE DEVELOPMENT OF VISCERAL CHANGES

By EMILE HOLMAN, M.D. (By Invitation)
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Speaking before this Association, in 1918, the late Professor Halsted discussed the enlargement of the heart that accompanies certain cases of arteriovenous aneurysm, and asserted that there must be some causative relationship between the two.[1] In 1919, again before this Association, Dr. Halsted reported a unique case of multiple congenital fistulæ between the external carotid artery and the veins of the neck, with marked dilatation of the vessels proximal to the fistulæ.[2] It was his stimulating speculation on the subject that aroused his pupils to investigate the problem, with the result that Reid[3] was able to present, in 1920, the first experimentally produced dilatation and hypertrophy of the heart which had developed as the result of an *A–V* fistula. However, it occurred only once in his series of 12 dogs, and no adequate explanation has been given until lately to account for its occurrence in certain cases and its absence in others. The duration of the fistula was emphasized as the main factor in its development.

In the past eighteen months I have had the opportunity to study 2 very instructive cases of arteriovenous aneurysm, the first a popliteal fistula of twenty-five years' duration, the second a femoral fistula of twenty-six years' duration, each case presenting a hugely dilated heart with an extraordinary enlargement also of the artery and vein proximal to the fistula.

A startling observation was made in Case I (Fig. 1) and again in Case II: Closure of the fistula by digital compression resulted in a remarkable transitory increase in general blood-pressure for a few beats, subsiding promptly to a level considerably higher than normal, where it remained as long as the fistula was closed. Opening the fistula caused the blood-pressure to drop to a very low point, with a prompt recovery to normal.

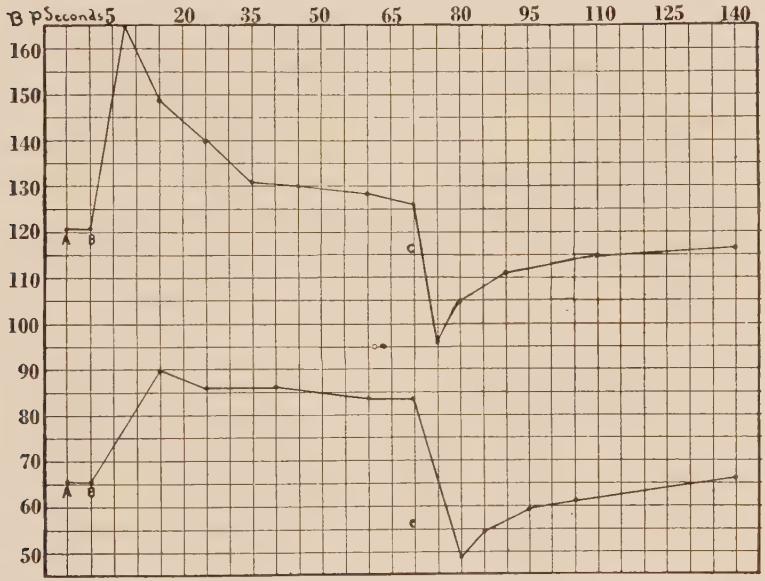

FIG. 1.—Blood-pressure changes in Case I on opening and closing the fistula. The lower line represents fluctuations in diastolic pressure; the upper, in systolic pressure. *A*, fistula open. *B*, fistula closed. *C*, fistula opened. (From Archives of Surgery, July, 1923.)

Case II (Fig. 2) illustrated this even more strikingly. Widely fluctuating changes in diastolic pressure paralleled those in systolic pressure in each instance. Accompanying the rise in pressure was a remarkable retardation in heart-beat, a pulse of 76 dropping to 40 and a pulse of 78 to 60, the low pulse persisting throughout the closure of the fistula.

The fistulous opening in each case was eliminated, the first

by repair, the second by excision, followed in each instance by a rapid subsidence of the dilated heart within three weeks after the operation.

Following the operations, there again occurred striking changes in blood-pressure: On the day immediately after the long three-

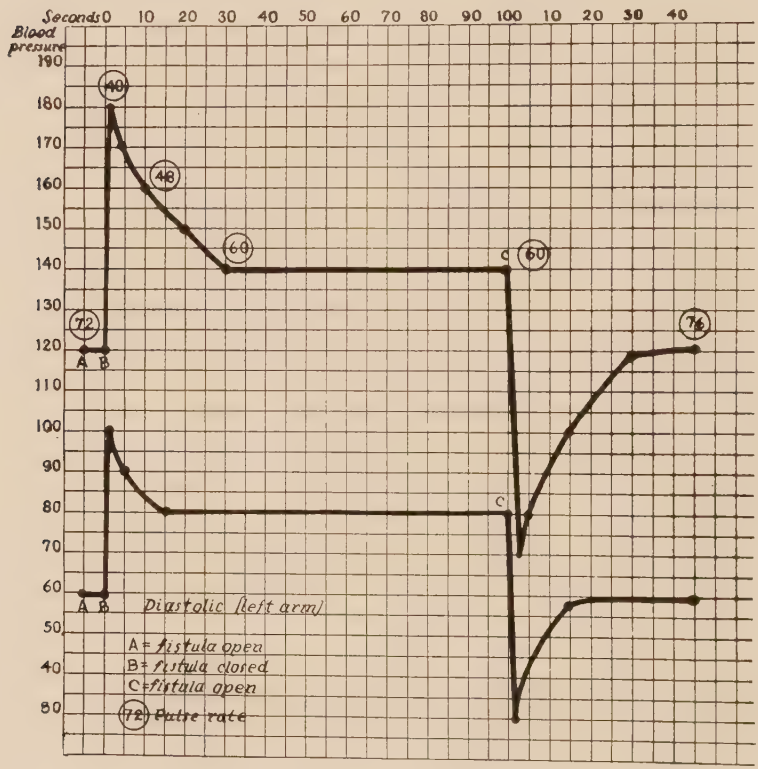

FIG. 2.—Blood-pressure changes in Case II on opening and closing the fistula.

and four-hour operations it was excessively increased, with a gradual subsidence during the next few days of the systolic pressure to normal, but with a permanent elevation of the diastolic level, resulting in a marked decrease in pulse pressure (Fig. 3). The pulse also showed a marked retardation during this period of high blood-pressure as illustrated in Case II (Fig. 4).

These startling variations in blood-pressure held our attention until an explanation was developed by applying the hydraulic

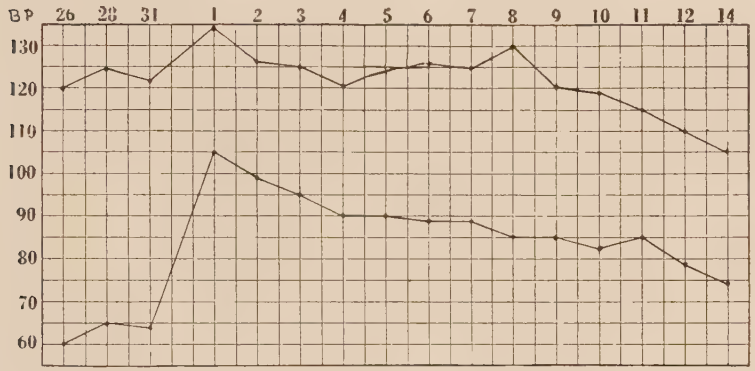

FIG. 3.—Blood-pressure changes in Case I following repair of the fistula on August 31, showing decreased pulse-pressure due to the elimination of the fistula. (From Archives of Surgery, July, 1923.)

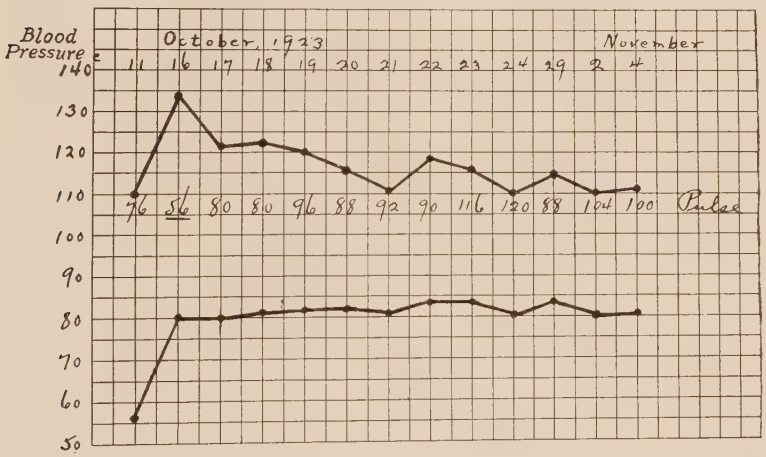

FIG. 4.—Blood-pressure and pulse changes in Case II following excision of the fistula on October 15. Note marked elevation of pressure and retardation of pulse on day following excision.

principles normally concerned in the maintenance of blood-pressure in the circulatory system. Physiologically, blood-pressure is controlled by the following variable factors:

1. Cardiac output or rate of contraction.
2. Total blood volume.
3. Total capacity of the system as determined by dilatation or contraction of the vascular bed.
4. Peripheral resistance.

Physiologists, and surgeons in particular, are acquainted with the tremendous changes in blood-pressure that may be effected by exsanguination, by vasodilator or vasoconstrictor drugs, as well as by hydremic plethora. The introduction of a fistula into the circulatory system results in a marked permanent reduction in peripheral resistance, with an accompanying effect on blood-pressure commensurate with this reduction in resistance. To compensate for the diminished resistance, the variable factors noted above come into play to maintain an adequate blood-pressure compatible with life.

The first compensatory adjustment is an increase in the rate of cardiac contraction. Experimentally, there is invariably an accelerated heart action in the presence of a fistula, particularly when the fistula is first opened. The size of the opening determines, of course, the extent of the diminished peripheral resistance, and a small fistula produces only slight and at the most a temporary acceleration of pulse. The effects of a large opening are typically illustrated by the following observations on Dog L28 (Fig. 5). A blood-pressure of 216/106 and a pulse of 84 were observed before operation. On October 22 an anastomosis, 2.5 cm. long, was established between the right common iliac vessels. A pulse-rate of 120 increased to 140 during the operation. On opening the fistula the pulse-rate rose immediately to 210, where it remained during the rest of that day. On the next day a blood-pressure of 110/60 and a pulse-rate of 200 were recorded. There then followed a gradual recovery of systolic opressure with an accompanying reduction in pulse-rate, but never to the preoperative rate. The chart illustrates the markedly increased pulse and pulse pressure in the presence of a fistula.

It is evident that some other factor besides an accelerated heart action was brought into play in this instance to reës-

tablish blood-pressure. Contraction of the vascular bed would be insignificant and at the most temporary in its effect. The only other variable factor is total blood volume. In a previous consideration of this problem[4] an increase in total volume was

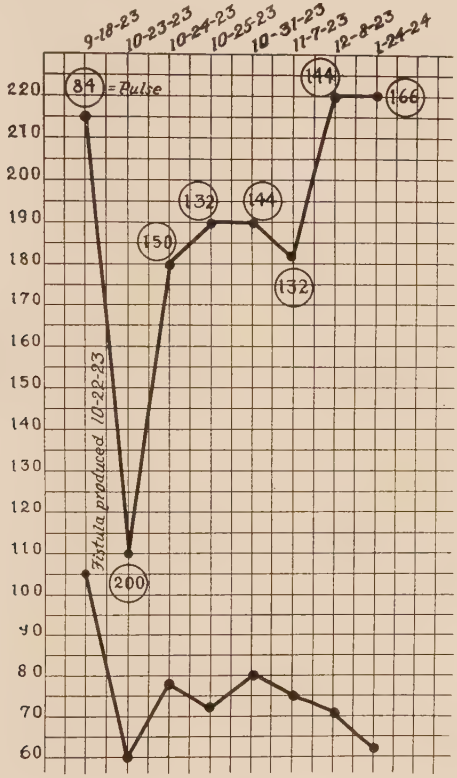

FIG. 5.—Chart to show pulse and blood-pressure changes in the presence of a large iliac fistula in Dog. L28. Marked fall in pressure and tremendous increase in pulse the day following production of the fistula.

postulated as an inevitable accompaniment of a fistula, which permits a large leakage of blood from the arterial into the venous systems. The experimental evidence available substantiates this hypothesis. It was contended that the changes incident to an arteriovenous fistula depended primarily on the amount of blood

short-circuited to the heart, and therefore on the size of the fistula (Fig. 6).

Dog L2, with a small femoral fistula, showed a blood volume of 1420 cc immediately before operation and a volume of 1400 cc five months later. About four weeks before the second reading was made the fistula had apparently undergone a spontaneous closure, as determined by the cessation of the thrill, due, we

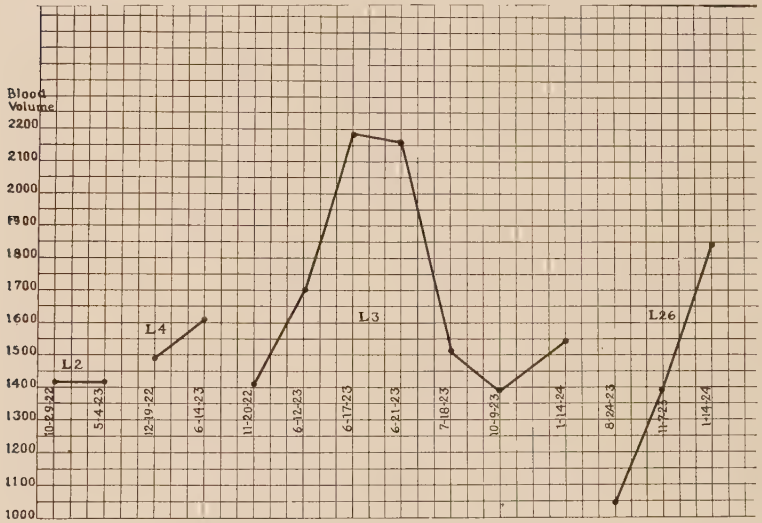

FIG. 6.—Blood volume variations in the presence of a fistula: Dog L2: small femoral fistula which closed spontaneously three months after its production. Dog L4: Small femoral fistula that peristed throughout life. Dog L3: Large femoral fistula with cardiac enlargement and blood-pressure changes on opening fistula. Dog. L26: Large iliac fistula with marked cardiac enlargement.

presume, to the small size of the fistula and the small amount of blood diverted through it. The spontaneous closure of small fistulæ was noted by Reid in 1920.

Dog L4, with a small fistula that persisted throughout life, showed a slight increase fom 1470 to 1630 cc in five months' time, with a slight detectable dilatation of the proximal vessels.

Dog L3, with a large femoral fistula, showed an increase in

six months' time from 1400 to 1680 cc, accompanied by cardiac enlargement and by blood-pressure changes on opening and closing the fistula. The fistula was excised, and on the second and fifth days following this excision, blood volume estimations of 2150 cc and 2130 cc were made. During this period of high blood volume the blood-pressure was markedly elevated to 250/125 and the pulse retarded to 60. Thirty days later the blood volume had dropped to 1500 cc, blood-pressure had subsided to normal, pulse had returned to a higher rate and the cardiac area had decreased in size. Four months later a blood-volume determination of 1385 cc was made, a second fistula between the iliac vessels was produced, and within a month blood volume had increased to 1530 cc with again an increase in cardiac area.

Dog L26, with a large iliac fistula, showed a blood volume of 1039 cc immediately before production of the fistula, 1340 cc three months later and 1840 cc in five months' time, accompanied by marked dilatation of the proximal vessels and considerable cardiac enlargement. During this time the dog also gained in weight from 12 to 15 kg., but there is still a marked relative increase in total blood volume, unexplained by the increase in weight.

The abnormal hydraulics resulting from a fistulous communication are better understood by applying the conception that a fistula establishes two systems of circulating blood (Fig. 7).

A. Heart-artery-capillary bed-vein.

B. Heart-artery-fistula-vein, each with a common source of supply, the heart, but with a great difference in peripheral resistance. Flowing water seeks the path of least resistance, and if it were mechanically possible the entire blood volume would be diverted through the fistula. It is obvious that if the peripheral resistance at point *B* is zero, and the peripheral resistance at *A* is considered to be the difference in pressure between the artery and vein at that point, or about 180 mm. mercury, a larger column of blood from the aorta will be directed toward the fistula with its lessened resistance than toward the capillary bed beyond *A*.

It is this increasing column of blood directed toward the fistula that acts as the distending force which dilates the feeding artery and the receiving vein. If the fistula is larger in cross-section than the feeding artery a larger and larger column of blood will be directed toward the artery leading to the fistula.

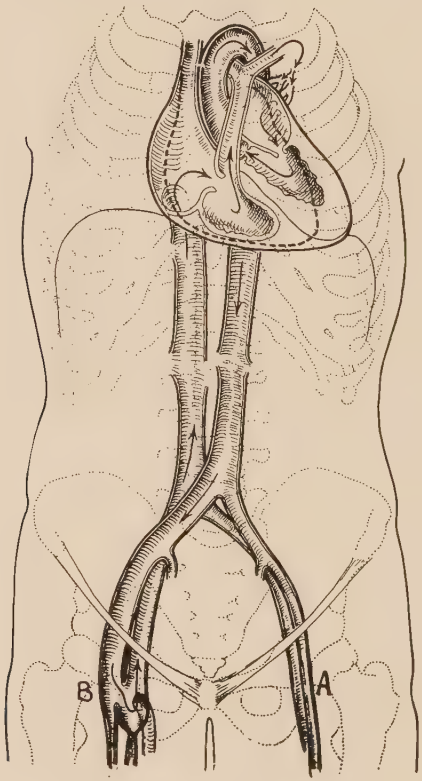

FIG. 7.—Schema of circulation in presence of the fistula: *A*, heart-artery-capillary bed-vein; *B*, heart-artery-fistula-vein.

The result is a gradually increasing volume of blood flowing through the fistula and back to the heart. This volume increase and the resulting dilatation of the vessels will be progressive until the resistance to an increase in flow through the fistula system *B* equals the resistance to the flow through the capillary

bed in *A*. The resistance in the fistula system depends upon (1) the size of the fistula, (2) the presence or absence of venous obstruction proximal to the fistula, (3) the limited distensibility of the arterial wall leading to the fistula, and (4) upon the resistance to dilatation furnished by the tissues and structures in which the artery and vein lie. Due to these factors, dilatation is a slowly progressive process, and while duration may not explain the dilatation, it does determine the extent of its development.

The gradual increase in the amount of blood short-circuited through the fistula increases, of course, the amount of blood flowing through the heart, and just as the vessels place themselves in harmony with the volume of blood traversing them, so the heart will dilate to accommodate this increased flow through its chambers.

It is suggested that the increasing total blood volume noted in our experiments is accommodated in this increasing capacity of the system heart-artery-fistula-vein. The blood-pressure and pulse changes noted clinically and experimentally on closing a fistula are then easily understood: Closure of the fistula results in forcing the large volume of blood, previously short-circuited through the fistula, into the normal capillary-bed circulation, all the vessels of which become distended by a volume of blood considerably increased over the normal. The elimination of the fistula eliminates likewise the area of diminished peripheral resistance. The result is an immediate increase in blood-pressure, and as a compensatory factor to offset the increased blood volume and increased peripheral resistance responsible for this increased pressure, an immediate retardation in pulse occurs. The permanent excision of the fistula, as we have seen, clinically and experimentally, produces a temporarily high blood-pressure and a retarded pulse. Coincident with a decreasing blood volume, this high blood-pressure falls and pulse-rate returns to normal.

Numerous experimental observations were made to illustrate these various points, only a few of which may be noted here

(Fig. 8). *X*-ray studies and autopsy findings indicated that small fistulæ resulted in no or only very slight dilatation of the heart or proximal vessels, nor did small fistulæ produce any blood-pressure or pulse changes on closing and opening the fistula. Large fistulæ were invariably accompanied by cardiac

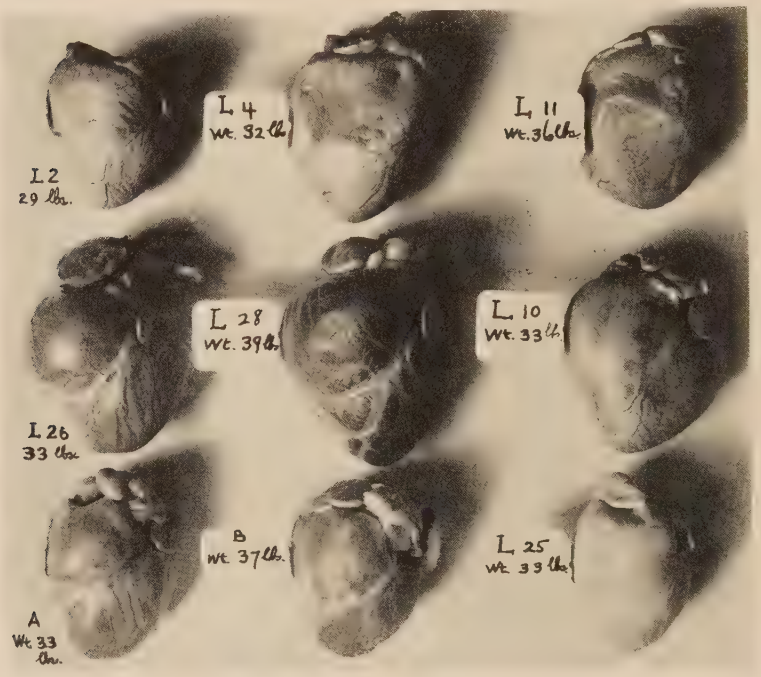

FIG. 8.—Hearts removed from dogs with fistulæ: L2 and L4 with small femoral fistulæ showed no enlargement. L11 with large femoral fistula but proximal vein ligated showed no enlargement. L26 and L28 with large iliac fistula showed great increase in size. *A*, and *B* normal hearts from normal dogs.

enlargement and dilatation of the proximal vessels except in Dog L11. In this instance the vein proximal to the large femoral fistula was ligated to prevent any free return flow to the heart, and neither the vessels nor heart showed any dilatation after three and a half months' time, and opening and closing the fistula

produced no blood-pressure changes. That the enlargement is due mainly to a dilatation is evident from the accompanying illustration (Fig. 9). The weights of the enlarged hearts revealed evidence of a definite cardiac hypertrophy, the result of the increased effort necessary to propel forward the increased flow through its chambers. Normal hearts weigh approximately 90 to 115 gm. in dogs weighing 12 to 15 kg. The hearts of dogs with large fistulæ weighed 130, 132, 144, 147, and 164 gm. The hearts of dogs with small fistulæ weighed 94, 103, and 106 gm.

FIG. 9.—Cardiac enlargement is mainly a dilatation.

This cardiac enlargement and the rapidity with which it may occur after the production of a fistula was well illustrated by Dog L19.

Fig. 10*a* shows the size of the dog's heart before the production of a fistula between the aorta and vena cava just above the trifurcation. Within seventeen days the heart dilated to an extraordinary degree (Fig. 10*b*), there was a marked increase in the vigor of cardiac effort, as indicated by the very tumultuous

heart action that could be palpated over the entire thoracic cage both in front and back. During the three weeks in which the opening was present the pulse was constantly around 180. The increasing cardiac area, the appearance of an increasing

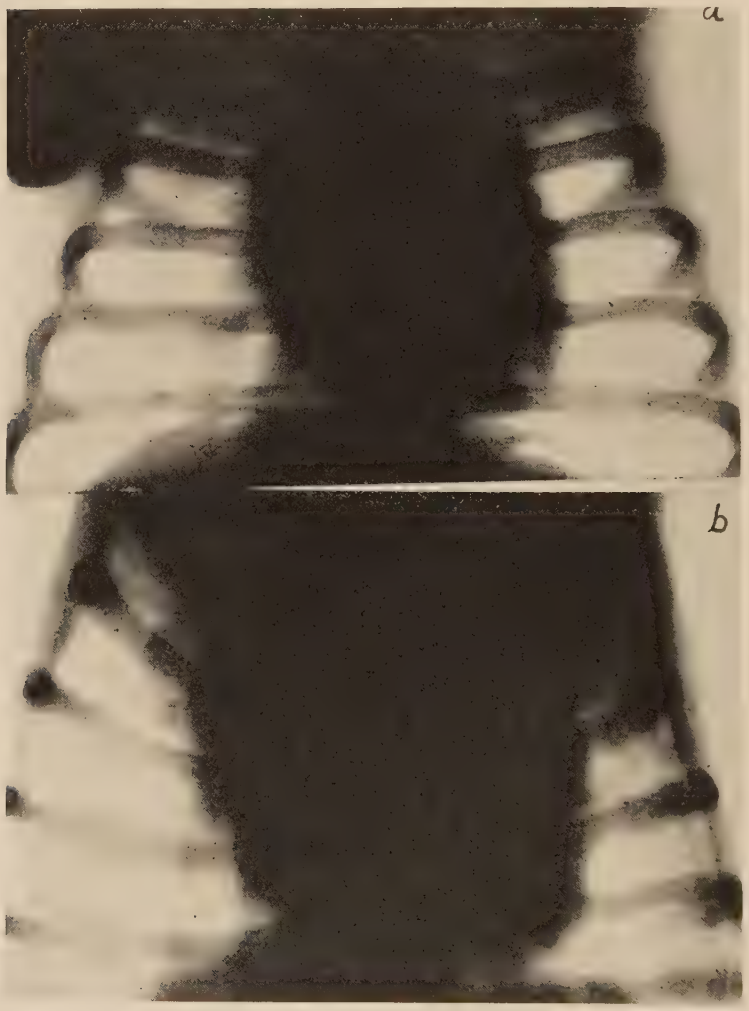

FIG. 10.—Cardiac areas in L19: *a* before production of a fistula between aorta and vena cava; *b* seventeen days after operation.

edema of the abdominal wall and the greatly accelerated and vigorous heart action suggested an early decompensation unless

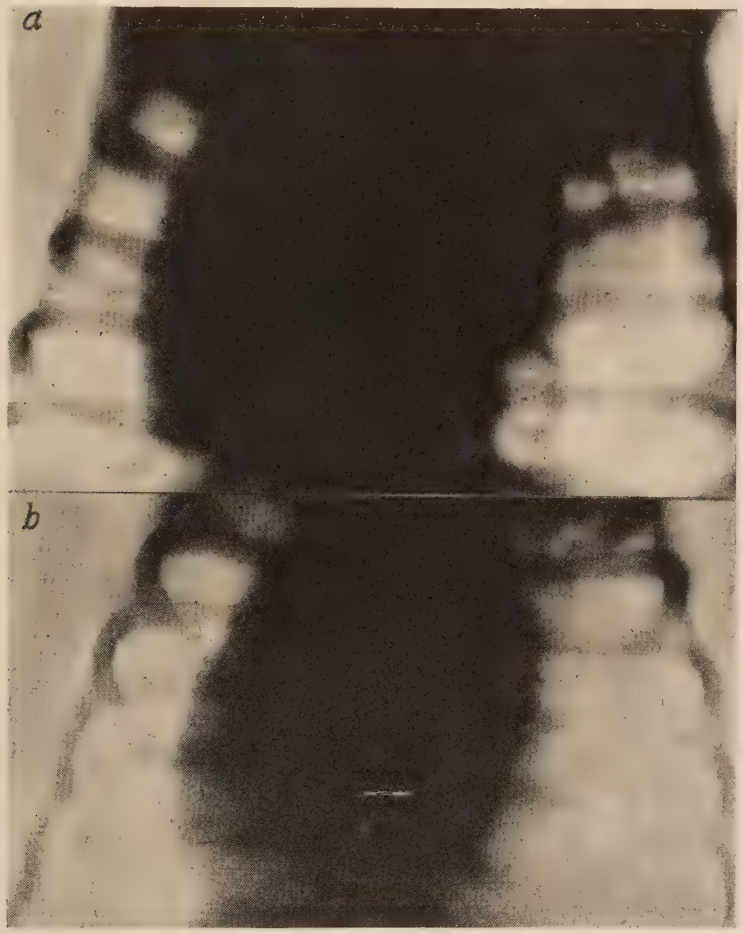

FIG. 11.—Cardiac areas in L19: *a* one day before and *b* three days after repair of the fistula.

the fistula were eliminated. Accordingly, by dint of a long four-hour operation, the aorta and vena cava were again separated and the normal channels restored. Within three days

after the repair of the fistula the heart had decreased markedly in size (Fig. 11), the pulse had subsided to 126 and the cardiac impulse could be felt as normally only over the anterior part of the thorax. This dog is still living.

The causative relationship between cardiac enlargement and arteriovenous fistula has been definitely established by these studies. It is attributed to an increasing volume of bloodflow through the system heart-artery-fistula-vein, an increased flow which is the direct result of the decreased peripheral resistance in this system as compared to that present in the normal capillary bed. Accompanying this increasing volume flow through the fistula there develops, *pari passu*, an increase in total blood volume as one of the compensatory adjustments for the short-circuited bloodflow, a necessary adjustment if general blood-pressure is to be maintained in the face of the marked fall in peripheral resistance. These adjustments depend upon the amount of blood diverted through the fistula, hence upon the size of the fistula and upon the absence of any obstruction to the free return flow of blood to the heart.

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CIRSOID ANEURYSM OF THE SCALP, WITH THE REPORT OF AN ADVANCED CASE

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ATLANTA, GA.

Presented by HARVEY CUSHING, M.D.
BOSTON, MASS.

Congenital telangiectases, vascular nevi or angiomas, which occur so commonly on the face and scalp, may be the starting point of disfiguring aneurysmal tumors. When the contributing arteries and the outgoing veins succeed in forming anastomoses through the cavernous bed of the nevus, the lesion becomes nothing more than a diffuse arteriovenous fistula. Such is the probable etiology of most so-called cirsoid aneurysms, in which the intermediary incidence of trauma in many cases seems definitely established.

In its mechanical effects, therefore, a cirsoid aneurysm resembles an arteriovenous fistula, with the difference that in the latter condition the communication is usually (though not invariably) single, whereas in the true cirsoid the communications are multiple and take place through the vascular meshes of the tumor.

The condition must have been recognized from the earliest times in view of its striking characteristics, and it would be an interesting study to trace in detail the early records of the lesion;[1] it is not at all improbable that some ancient example may have given rise to the legend of the serpents in the hair of

[1] For anyone so disposed, the section in Rudolf Virchow's classical treatise on tumors (Die Krankhaften Geschwülste, iii, 471 *et seq.*) would make a proper point of departure.

Medusa, which Perseus cured by radical though unprofessional measures.

It would appear that the term *anévrysme cirsoïde* (varix-like) was first used by Brescht in 1833, long after William Hunter's description of "aneurysm by anastomosis." Though other designations were introduced, as *aneurysma serpentinum*, by Cruveilhier, the term "cirsoid", accepted by Verneuil and by A. Robert in 1851, as well as by Ch. Rubin in his excellent description of the erectile tumors three years later, has subsequently come to be the one generally employed in French and English literature.

In the German literature, however, the cases, for the most part must be sought under other titles, due largely to the influence of Virchow, who proposed the term *aneurysma racemosum* or *ranken aneurysma*. Moreover, fully cognizant of the fact that a congenital angioma was often the starting point of these lesions, he designated them as *rankenangiom* or *angioma racemosum arteriale*.

Undoubtedly the cases in the early literature, many of which are cited by Virchow, comprise lesions of various kinds, not all of them in association with primary angiomas, for it is quite probable that arteriovenous fistulæ (more especially those with single or multiple communications involving the vessels of the face and neck) may arise through some faulty vascular development which may leave one or more arteriovenous communications without the intermediation of a cavernous angioma.[1]

From this preamble it may be gathered that there are several varieties of so-called cirsoid aneurysm arising in various parts of the body which deserve clinical and pathological differentiation.

[1] It is quite possible that the recent case reported by F. L. Meleney (Surg., Gynec. and Obst., 1923, xxxvi, 547) was of this type. The patient had a small arteriovenous aneurysm under the chin. Dr. Meleney has supplied "a complete bibliography of the literature as given in the Index Medicus since 1879". This bibliography, however, is confined almost entirely to articles which have appeared under the title of "cirsoid aneurysms", whereas almost an equal number of important articles, chiefly in the German literature and otherwise entitled, do not appear in his list.

The present communication, however, will be limited solely to a brief consideration of the anastomosing aneurysms of one type and in one situation: namely, those which affect the scalp and which arise through the intermediation of a preëxisting vascular abnormality of a congenital, angiomatous nature.

In any outspoken example of this condition the clinical picture is such a startling one that the case is likely to be reported. The extensive literature on the subject shows that these cirsoid aneurysms more often occur on the face and head, whether for the reason that congenital vascular nevi are more common there, or because the head is more exposed to the kind of trauma likely to provoke an aneurysmal change in the preëxisting lesion, is not apparent. Nevertheless, cirsoid aneurysms of the advanced type observed in the patient whose case-history follows are not common. It is the first example of the condition in the *circa* among 35,000 admissions to the Brigham Hospital.

P. B. B. H., Surgical No. 17539. John A., an Italian wool-worker, aged twenty-two years, entered the medical service of the Brigham Hospital, October 9, 1922, because of a chronic cough, "swellings" on his forehead and headaches. Though suspected of having incipient pulmonary tuberculosis, he was soon transferred to the surgical service for treatment of his aneurysm.

In 1918, while serving in the Italian Army, he had received some minor shrapnel wounds of the scalp, and was subsequently taken prisoner. His wounds, which were confined to the occipital region, required no especial care, and though none of them appear to have affected the central region of the aneurysm, he states that the top of his head was badly contused at the time.

To this trauma he naturally ascribes the present condition, for soon afterward he first noticed the pulsating swelling under the frontal hair margin, which has gradually increased in size. He soon began to be conscious of a roaring sound in his ears, and at present this bruit and the startling appearance of the huge vein in his forehead constitute his chief annoyances.

Examination showed a vigorous, alert, young Italian who, aside from a chronic bronchitis of doubtful origin, appeared to be in good general physical condition. The outstanding feature of his case lay in the lesion, thus described by Dr. Cushing in a note dictated shortly before the operation, when the accompanying photographs were taken (Figs. 1, 2, 3).

"Before this man's head was shaved there was discernible through the hair a faintly reddish, soft, warm, pulsating tumor measuring approximately 6 by 6 cm., slightly elevated above the surface of the rest of the scalp and situated in the midline about at the coronal suture. Radiating from this tumor in all directions, but chiefly laterally and anteriorly, are huge, tortuous vessels, the appearance of which naturally suggests the snakes in the head of Medusa. Through the hair the slightly prominent central area, owing to its discoloration and increased heat, gave the impression of an inflammatory process on the verge of ulceration. After shaving, however, it becomes evident that the central lesion is a reddish nevus, doubtless congenital, of the port-wine mark variety, which overlies a cavernous angioma.

"The enlarged and tortuous vessels which radiate from the lesion are chiefly confined to the anterior half of the scalp. The three main outstanding channels are evidently veins, though they show visible pulsation and have a palpable thrill. The largest of these is a broad vessel measuring nearly 4 cm. at its widest point, which passes downward toward the root of the nose. There it bifurcates and extends on each side as far as the alæ. Another large tortuous vein passes off from the right side of the central swelling and extends down toward the anterior part of the ear, where, like the aforementioned vessel, it becomes gradually narrower and finally disappears. There are similar vessels on the left side of the scalp, though less marked than those described.

"These vessels, which are obviously huge, pulsating, venous channels, are easily distinguished from the arteries, which, however, are likewise unduly prominent and unusually tortuous,

and which can be palpated all the way from their points of appearance on the scalp directly into tumor. This is particularly true of the greatly dilated anterior branch of the right temporal, which can be traced directly into the site of the main central

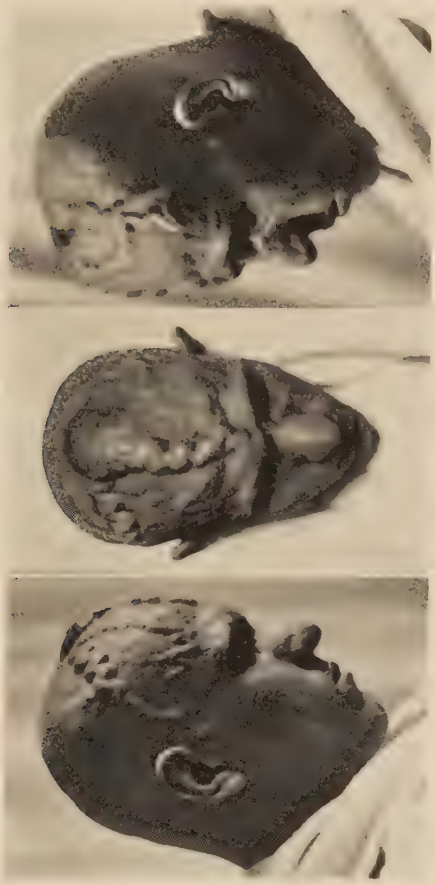

FIG. 1 FIG. 2 FIG. 3

FIGS. 1, 2, 3.—Preoperative condition, October 18, 1922.

lesion; the same is true of the left temporal as well as the supra-orbital arteries. The suboccipital arteries are likewise full and large, and pulsating vessels can be felt over the scalp in the occipital region as well as in the anterior areas, where, however, they are particularly pronounced.

"While the patient is sitting up it is possible to empty the large vessels by pressing upon them near their point of emergence from the main central lesion, proving that they are efferent veins. Moreover, they are visibly pulsating, and there can be no question, therefore, but that arterial circulation, through some form of communication, is thrown directly into them. The central tumor or swelling is pulsating and apparently contains a large lake of blood. Its tension varies considerably, depending upon the position of the patient; but even when he is sitting up and when all the arteries possible are compressed, it still remains tense and pulsating.

"Pretty much over the entire scalp is heard an astounding bruit, which is audible to the patient and which is precisely like that accompanying an arterial venous aneurysm. It is curious, however, that this bruit, though heard all over the head, is louder at the lower margins of the scalp, particularly in the temporal region and over the glabella, than it is directly over the pulsating tumor itself, where, as a matter of fact, the bruit is scarcely to be heard.

"The *x*-ray plates show greatly dilated sphenoparietal grooves, and it is quite probable that there is a dilatation of the meningeal vessels corresponding to that of the extracranial vessels. The meningeal artery consequently may participate in the process, and it is possible that the dilated meningeals may communicate with the longitudinal sinus, and thus with the extracranial pulsating mass which has been described. This seems the more probable in view of the fact that when a tourniquet is applied around the head, shutting off the extracranial arterial supply, there is an enormous increase in the size of the veins of the 'caput,' which distend to such a degree that rupture seems impending. This observation makes it appear that a bilateral ligation of the external carotids will be a necessary preliminary step to the operation."

Dr. Cushing's operative note, November 4, 1922, is in large part as follows:

"*Ligation of each external carotid artery. Reflection of scalp, disclosing cavernous angioma. Puckering of angioma by multiple sutures. Replacement and suture of scalp in layers.*

"The external carotids were exposed, first on the right side, where the vessel was immediately ligated; then on the left, where a provisional ligature was thrown around the vessel and a bulldog clip applied, so that the circulation might be reëstablished should a slough of the scalp be threatened.

"A horseshoe-shaped incision with its base posteriorly, was then made around the central pulsating angiomatous mass. By the usual method of finger compression of the scalp this incision was carried down through the galea and the huge vessels at each side were caught by clamps. Naturally there was no arterial bleeding, but the large veins remained as full apparently as before the carotid ligations. On reflecting the flap of the scalp in the subaponeurotic layer the central angiomatous tumor was exposed. It seemed to lie more or less within its own pericranial capsule, from which the scalp could be dissected away without great difficulty. Fortunately, this central lesion had a great deal of fibrous tissue, so that it could be caught by a series of heavy curved clamps as the scalp was peeled away from it. Subsequently a multitude of through-and-through silk sutures were taken where the tumor had been gathered up in these clamps, and thus the growth was thoroughly 'puckered' in the hope that thrombosis thereby might be encouraged.

"There was no special difficulty in this procedure and the hemostasis was fairly complete. The scalp was then replaced and closed carefully in layers, as usual, without any special attention being paid to the individual dilated vascular channels. The bulldog clip was then removed from the left external carotid, but the arteries in the scalp promptly filled, and it was evident that there would be some bleeding. The provisional ligature, therefore, was tied before closing the wound in the neck."

The circulation in the scalp seemed perfectly good when the usual dressing was applied. However, a very slight superficial slough of the flap margins occurred as the broad scars in the

accompanying photographs (Figs. 4, 5, 6) make evident. The central lesion was subsequently radiated to encourage endothelial destruction and thrombosis.

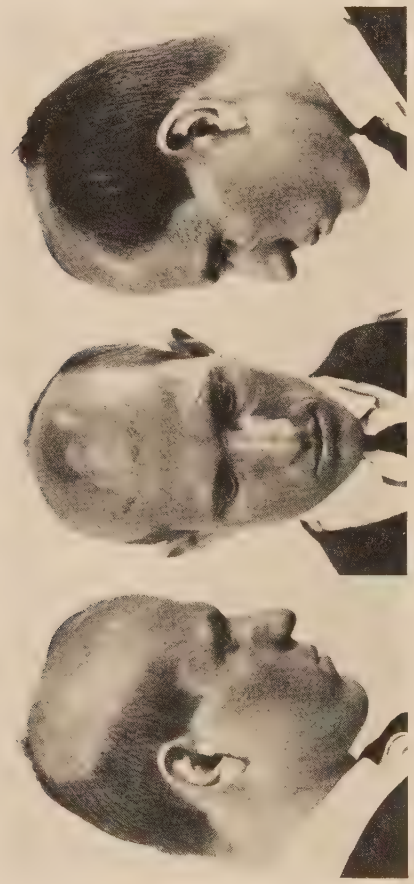

FIG. 6

FIG. 5

FIG. 4

FIGS. 4, 5, 6.—Condition February 2, 1923, three months after operation, following radiotherapy.

As is evident from this description, the operation consisted in an attempt to interrupt the vascular channels by an incision encircling the central lesion, in the hope of producing thromboses which would cure the fistulous character of the lesion without removing the benign angioma itself. The operation was less formidable than anticipated, and it is possible that the

cavernous portion of the angioma underlying the galea might have been subsequently removed. This would have meant carrying the dissection down to the skull and removing the tumor together with the pericranium, but, as stated in Dr. Cushing's

FIG. 7 FIG. 8 FIG. 9

FIGS. 7, 8, 9.—Condition one year after operation, February 28, 1924.

note, it was assumed that the lesion had vascular connections through the skull with the sinus longitudinalis, and was possibly fed by the meningeal vessels. The outcome of the operation showed that this was apparently a mistaken idea; and though the central angioma, therefore, might have been removed, it is

an innocent vascular anomaly in which the possible acquirement of further aneurysmal characteristics seems remote. The lesion remains quiescent at the present writing, a year after the operation (Figs. 7, 8, 9).

COMMENT. As stated in the introductory paragraphs, it is the purpose of this paper merely to put on record another outspoken example of cirsoid aneurysm of the scalp with no pretence of covering the full literature of the subject. A large number of these cases have been recorded, and many of them have been successfully treated, either with amelioration or with cure, by a great variety of surgical procedures: by single or bilateral carotid ligations; by multiple circumferential ligations; by attempts to obliterate the lesion with the galvanocautery; by acupressure; by the injection of thrombosing or scar-forming substances; or, indeed, by extirpation.

Of the many remarkable examples in the literature, possibly the case best known to surgeons was that reported by H. Müller,[1] in 1891, from the clinic of Paul Bruns. His paper was accompanied by a drawing which has passed down through several generations of texts and has consequently familiarized many with the appearances of the lesion on postmortem dissection. As is so common, the patient had a red birthmark at the hair margin of his forehead which greatly increased in size and by his twentieth year had acquired the appearances shown in the dissection. Bruns first ligated the right external carotid, and on attempting the same thing on the other side encountered severe bleeding and was compelled to ligate the left common carotid. Hemiplegia followed with a fatality and Ziegler made the studies which have given us this striking illustration of the lesion.

To be sure, many other examples occur in the German literature, the surgical aspects of which are chiefly emphasized, some of them representing very formidable conditions. An excellent

[1] Ein Fall von arteriellem Rankenangiom des Kopfes, Beitr. z. klin. Chir., 1891, viii, 79–91.

résumé of the subject up to 1883 was given by Hermann Kümmell.[1] One of the better-known cases subsequently reported is that by Paul Clairmont,[2] in 1908, the lesion having been attacked in two stages without preliminary carotid ligation. Another outspoken case, reported by Max Kepler,[3] was operated upon by Heineke in 1910: a huge cavernous cirsoid of the parietoöccipital region, a very desperate and bloody procedure carried out in two stages, with a subsequent skingraft.

The French literature likewise contains a good many comparable examples, and recently Noordenbos, of Amsterdam, has reported a case[4] very similar to the one the subject of this report. At the age of twelve the patient had applied for removal of a small vascular tumor on his forehead, but operation was thought inadvisable. Twenty years later, because of its enormous increase in size and its tendency to bleed, intervention was imperative. At operation the external carotid and frontal arteries were ligated on both sides. Catgut sutures were then passed around each afferent vessel and tied over a small roll of gauze. The whole tumor mass was then excised down to the periosteum. A subsequent skingraft resulted in complete recovery.

The question of an intracranial extension of the lesion, which was feared in the case herein reported, has arisen in the minds of a good many others, and has been thought probable in view of the fact that in a number of patients (*e. g.*, Clairmont's) epilepsy has supervened or else there have been definite evidences of mental deterioration. Indeed, the question was raised as long ago as by Guérin (1870) as to whether an extensive cirsoid of the scalp could come into communication with the intracranial vessels and sinuses. It would appear, however, as was true of our patient, that there was no real ground for this apprehension,

[1] Zur Behandlung des Angioma arteriale racemosum, Arch. f. klin. Chir. 1883, xxviii, 194–213.

[2] Ibid., 1908, lxxxv, 549.

[3] Zur Behandlung des Aneurysma arteriale racemosum, Beitr. z. klin. Chir., 1912, lxxviii, 521–536.

[4] Noordenbos, W., and de Jong, R.: Aneurysma Arteriale Racemosum van het Hoofd, Nederl. Tijdsch. v. Geneesk., October 12, 1918, ii, 1224–1235.

and seemingly in all other cases, in spite of the apparent grooving of the skull by the huge vessels, operation has disclosed no alteration in the skull or evidence of intracranial communication. There nevertheless have been reported a good many examples of aneurysmal varices which have originated within the cranial chamber. This, however, is quite another subject.

Very little has been said in the foregoing paragraphs of the cases which have been published from American clinics—all of them, to be sure, very briefly recorded. The older medical literature antedating the great medical indices, if thoroughly perused, would of course be found to contain the records of some of these surprising lesions. Attention may be drawn to two of these early reports at least. One of them was put on record in 1853 by Dr. R. D. Mussey,[1] professor of operative surgery in the Miami Medical College, at Cincinnati, Ohio, who described his case under the caption of "Aneurysmal Tumors upon the Ear Treated by Ligation of both Carotids." He speaks of the lesion as being "like an aneurysmal varix, which had arisen in a congenital nevus."

Another example will be found in a volume published in 1867 by J. Mason Warren,[2] then surgeon to the Massachusetts General Hospital. This case, similar to our own, was described as a so-called cirsoid aneurysm of the scalp arising from a congenital nevus starting about at the midline in the roots of the hair, "which gave him a very formidable aspect". And Dr. Warren goes on at length to tell of the many and varied procedures which were carried out by ligatures thrown over needles, followed by the applications of caustic potash repeated between twenty and thirty times to destroy the central lesion.

There are doubtless other similar cases buried in the early American literature. The following list, which may be incomplete, will nevertheless serve to assemble most of the cirsoid

[1] Am. J. M. Sc., 1853, xxvi, 333.

[2] Surgical Observations, with Cases and Operations. Ticknor & Fields, Boston, 1867. Case CCLXII, 451.

aneurysms of the scalp which, since 1887, have been put on record in this country:

1. T. M. Markoe, of New York (*Philadelphia Med. News*, 1887, l, 270): A brief report of a case following trauma, with ligation of both external carotids and probable improvement.

2. Herman Mynter, of Buffalo (*Ann. Surg.*, 1890, xi, 93): A case with ligation of one external carotid followed by circumferential ligation of vessels. Improvement, but no late report.

3. Willy Meyer, of New York (*N. York M. J.*, 1892, lvi, 214): A very brief report, possibly of an arteriovenous fistula rather than a true cirsoid aneurysm involving the temporal region. Primary ligation of external carotid with excision of aneurysm. In the discussion cases were mentioned by J. D. Bryant and Kammerer.

4. William D. Hamilton, of Columbus, Ohio (*N. York M. J.*, 1894, lx, 550): Ligation of one common carotid. Improvement, but no late report.

5. W. S. Forbes, of Philadelphia (*Philadelphia Med. News*, xlvi, 663): A case almost exactly like our own, with central pulsating tumor following trauma. Treatment, acupressure of the entering vessels and subsequent excision of central tumor. Recovery.

6. William B. Coley, of New York (*Ann. Surg.*, 1901, xxxiv, 414): Unilateral case with involvement of the right temporal region; not advanced. Ligation of the right external carotid followed by direct attack on tumor. "Nearly 100 ligatures were applied." Recovery, but result uncertain.

7. Carl Beck, of New York (*Ann. Surg.*, 1903, xxxviii, 496): "Aggravated case" following trauma. Ligation of temporal and other arteries, with extirpation of tumor. Profuse hemorrhage. Recovery.

8. Rudolph Matas, of New Orleans (*N. Orl. M. & S. J.*, 1908–9, lxi, 469): Brief report at a society meeting; probably a true post-traumatic arteriovenous fistula; not a cirsoid case, though so reported. A bilateral, pulsating exophthalmos was present following fracture of the base of the skull. Ligation

of both external carotids. Temporary relief with slowly progressing recurrence.

9. E. S. Judd, of the Mayo Clinic (*St. Paul M. J.*, 1916, xviii, 48): Perhaps the most extreme example of the condition on record. Ligation of both external carotids, followed a week later by bilateral reflection of the scalp and dissection of vessels. Probably complete cure.

DISCUSSION ON THE PAPERS OF DRS. LYLE, GIBBON, MATAS, RANKIN, CHARLES A. PORTER, BROOKS, HOLMAN ELKIN AND CUSHING.

Sir D'Arcy Power, K.B.E., London, Eng.:

I am only a straggler on the outskirts of this noble army of surgeons, but stragglers sometimes have the opportunity of making interesting discoveries. We are not accustomed to think of the great poets of the world as being interested in the surgery of the vascular system. I show you, therefore, this note-book, which contains a summary of the lectures attended by John Keats at the United Borough Hospitals in London, when he was a student of medicine. You will see from the page which is shown on the screen that it is in his own handwriting and that "Mr. C.," who was afterward Sir Astley Cooper, was lecturing upon "Aneurysm." The original note-book is preserved in the Hampstead General Library in London, and was formerly in the possession of Sir Charles Dilke. Four copies of it have been rotagraphed, and this one, which is sent to the Harvard University by Sir William Hale-White, I had bound by my sister, Mrs. Looseley. You will remember that Keats actually graduated and practised medicine for a short time, but he soon devoted himself entirely to poetry.

Dr. Evarts A. Graham, St. Louis, Mo.:

I wish to speak only in regard to this presentation of Dr. Brooks. I have been tremendously impressed watching Dr. Brooks at work on these cases, and with the very obvious practical significance of this work in addition to the more theoretical aspect. As a matter of practical diagnosis in a great many cases, his work has been of the utmost value. I would refer particularly to the case of a man with gangrene of all the toes on one foot. By utilizing the method which he has explained to you this morning, it was possible to show that there was no interference whatever to the arterial supply to that foot above the gangrenous portion, and in all probablity the gangrene had resulted from the wearing continuously of shoes that were too tight. The application of the method in this case saved this man from what undoubtedly in the hands of most surgeons would have been an amputation higher up.

The case of the diabetic who had an ulcer and was saved from the necessity of an extensive amputation because it was perfectly possible to show that the ulcer was due to a burn associated with an anesthesia of his foot rather than from serious vascular disturbance, also illus-

trated the practical value of the procedure. Even these 2 cases, I think, show a great triumph in diagnosis for this very ingenious method as explained to you this morning.

Dr. Ellsworth Eliot, Jr., New York:

I desire particularly to discuss the papers dealing with aneurysm. I think it is well to divide this subject into three general groups: (1) Aneurysm in the extremities in which the collateral circulation is quite adequate to maintain the circulation of the extremity after excision. A case of this kind is reported by Dr. Lyle. I have had a similar experience in an aneurysm of the radial artery just before it passes between the heads of the abductor indicis. We have all seen traumatic aneurysms of the scalp in which excision has yielded most satisfactory results. (2) Aneurysm of the extremities in which the question of the establishment of a satisfactory collateral circulation after excision cannot be definitely decided. In this group the Matas operation is unquestionably the operation of choice if done by skilful hands. Gangrene and secondary hemorrhage, sequelæ to be feared after any operation for aneurysm, are doubtless more common where the Matas operation is done by inexperienced surgeons. (3) Aneurysms of the root of the neck, including innominate aneurysm, comprise a third group. In 1912 I reported before this Association a case of ligation of the first part of the right subclavian artery for aneurysm of the third portion. This patient was completely cured, and when last seen, six years after the operation, was free from recurrence. From this and other results I am inclined to the selection of some form of ligation for the treatment of these cases. I would like to hear from Dr. Matas, as he closes the discussion, whether he shares this view or whether he believes that his operation is of greater service in this class of cases, and in the latter event what results have been secured.

There is one further clinical symptom that I would like to emphasize in connection with the subject of aneurysmal varix. In these cases, even where the aneurysm is of small size, the characteristic to-and-fro murmur may be transmitted a considerable distance along the course of the artery. I recall one instance, in a young man, of a small pulsating tumor in the thigh following a stab wound, in which the to-and-fro murmur was transmitted down to the ankle. The tumor, no larger than a marble, proved to be an aneurysmal varix of the anastomotica artery and vein and was successfully excised without damage to the femoral vessels. From the intensity of the murmur and the distance to which it was transmitted it was thought likely that the aneurysm involved the femoral vessels rather than one of their branches.

Dr. Joseph A. Blake, New York:

In connection with Dr. Cushing's paper on "Cirsoid Aneurysm of the Scalp," two weeks ago a patient came to see me at my clinic who was seventy years of age. In 1904 or 1905 I had removed a cirsoid aneurysm from this patient at the Roosevelt Hospital. The aneurysm occupied the whole left side of the forehead, reached up to the hair line and went across to the right side. Operation was carried out by ligating the temporals on each side and then gradually carrying the incision across and ligating all the vessels as encountered, with a final removal of the entire aneurysm *en masse*. He presented himself two weeks ago, having been perfectly well in the interval until a year ago, when a small cirsoid aneurysm appeared above the center of the forehead, and which at present is about 4 cm. in diameter. It is now nearly twenty years since the first operation.

I should like also to report a case of arteriovenous aneurysm which I think is of clinical interest. I operated upon this man in the winter of 1916, in France, for an arteriovenous aneurysm resulting from a bullet wound going between the internal jugular and the carotid on the right side. I hesitated for a long time to operate, but finally decided to do so. Just as I was starting the operation my assistant was called away to a case in collapse, so that I had to proceed with only a student assistant. Although the aneurysmal sac did not seem to be extensive, in my dissection I ruptured into it, with resulting serious hemorrhage. I had to control the hemorrhage by pressure with the one hand and pass the ligatures around the vessels with the other, my assistant not being of much help. I finally had to ligate the vessels *en masse*, as I could not dissect them apart, because I could not release my grasp controlling the hemorrhage. I passed two ligatures, one above and one below, including both the internal jugular and carotid, and also the recurrent laryngeal nerve. The man made a good recovery. He was a singer, and I hoped that the pressure upon the nerve was not sufficient to cause entire destruction of his voice. Much to my satisfaction he regained his voice in four months.

Dr. Alexis V. Moschcowitz, New York:

In connection with these papers I wish to report that a few years ago I was able to watch the formation of an aneurysm in a case that was a comparatively simple one. About five years ago there was admitted to my service a man with an extensive phlegmon of the hand and forearm. Numerous incisions were made and the case subsequently treated by the Carrel-Dakin method. Just prior to his

discharge from the hospital into ambulatory treatment, with superficial granulating wounds, I examined the hand and saw that the radial artery, just where it was crossed by a Carrel tube, was the site of aneurysm about the size of a large pea. This was excised and the patient discharged. My associate, Dr. Wilensky, will publish this case shortly in connection with other cases of trauma to vessels.

Dr. Leonard Freeman, Denver, Colo.:

With reference to Dr. Matas' paper, in which he refers to the return of the circulation through the aorta after ligation of the aorta with tape, I had a similar experience with the carotid artery in ligating with tape, except that in the carotid artery I used fascia lata instead of cotton tape. Fascia, as you know, does not stretch, so that Dr. Matas' assumption that the return of circulation was due to the stretching of the cotton tape would hardly apply to the fascial tape, especially as I wrapped the tape twice around the carotid artery. Dr. Perthes, who has done many ligations with fascia, has had a similar experience with leakage, and he asserts that it can only be prevented by applying another ligature distal to the fascial ligature.

It would seem that this is a very uncertain method of stopping the circulation through a vessel, the reason being that the tape does not injure the internal coat of the vessel, and when this is not injured an occluding thrombus does not occur. It is thus placed somewhat in the same category as ligating the pyloric orifice of the stomach, which is notoriously a difficult procedure to carry out effectually.

There are two theories about the development of cerebral symptoms after ligating the carotid artery—one being that the symptoms are due to anemia and the other that they are due to thrombosis and embolism. There is a good deal to support the thrombosis and embolism theory, the principal reason being that the symptoms do not occur at once, as they should if due to anemia, but often are delayed for several hours, days or even weeks.

I believe that in ligating the carotid artery or any other large vessels, we should always apply an ordinary ligature distal to the point of ligation in order effectually to control the circulation. It is interesting to note that Perthes was able to obtain more reliable results by placing a pad of folded fascia beneath the primary fasical ligature.

Dr. Vilray P. Blair, St. Louis, Mo.:

When the question was brought up of compression of the innominate artery, Dr. Graham asked me to take care of a man who had an aneurysm of the right carotid. Under local anesthesia the innominate

was exposed just where it comes out of the aorta and compressed and then the aneurysm attacked. A good deal of blood was lost and the man died on the table. I think it was from shock.

Dr. James Morley Hitzrot, New York:

I should like to confirm Dr. Gibbon's statement with reference to the greater efficacy of iodide of potash as compared to salvarsan in peripheral aneurysm with a syphilitic background. In a young colored man with a popliteal aneurysm thoroughly treated by salvarsan the aneurysm contined to increase. When iodide in increasing doses was substituted the growth ceased and possibly diminished slightly. I subsequently did an obliterative endoaneurysmorrhaphy (Matas), with a successful result.

Dr. John F. Connors, New York:

I would like to add a few words about Dr. Gibbon's paper. I agree with Dr. Gibbon that the operation of selection in these cases is the one of repair rather than that of ligation. Previous to my experience in France, I treated arteriovenous aneurysms by the ligation method, and I was led to adopt Dr. Matas' operation by an experience in a case in which I removed a small piece of high explosive from the common carotid artery, and I was amazed at the fact that no bleeding followed its removal. In 1920 I reported 10 cases with 1 death, the cause of death being an infection following the repair of a popliteal arteriovenous aneurysm. Since 1920 I have done four operations for repair of arteriovenous aneurysm, with 2 deaths, 1 of the femoral artery and 1 of the popliteal. These 2 deaths were due to infection. In both these cases a drain was used, and I feel that Dr. Gibbon is right when he says that no drainage should be used. In the 3 deaths which I have had, drainage was employed. I also agree with Dr. Gibbon that no clamp should be used. I used a piece of catgut, with which I angulated the artery to control the bleeding. One can readily release this piece of catgut to test the closure, and if bleeding occurs the circulation can be stopped very readily.

I do not agree with Dr. Eliot when he says that this operation can only be done under the most advantageous conditions, and I believe that if more of us would try this operation of repair, as described by Dr. Matas, the less frequently ligation will be attempted.

Dr. Carl A. Hamann, Cleveland, Ohio:

I wish, in this connection, to refer to the reopening of the artery after ligation in a patient in whom we tied the abdominal aorta with

a single heavy braided silk ligature. The autopsy, six months later, showed that there was a partial reëstablishment of the lumen.

With regard to the method of studying the circulation, Dr. G. N. Stewart has devised a method of estimating the bloodflow on the two sides. Such a method might be of use in determining the advisability of operation in cases of gangrene or impending gangrene.

Dr. Randolph Winslow, Baltimore, Md.:

With reference to the paper of Dr. Lyle, I had many years ago a case of the same character in which I ligated both the radial and ulnar arteries at the wrist, with a cure of the case. I cannot see the advantage of making a big scar in the palm of the hand and of running the risk of injuring the nerves, tendons and other structures when it is a perfectly simple operation to make a little incision on each side of the forearm, which, as far as I can see, answers the same purpose.

Dr. J. Shelton Horsley, Richmond, Va.:

Experimental arterial suturing is quite different from arterial suturing in old aneurysms, particularly in old arteriovenous aneurysms. The technic used in dogs would be quite the same thing as that used in a fresh wound in an artery in a patient a few hours after the injury, but on account of the dense scar tissue present the fine needles and thread employed in experimental work cannot be used in old arteriovenous or arterial aneurysms.

There is much more tendency for arteries to open, even in the smaller vessels, in the human being than in the dog. I have had 2 cases of small traumatic aneurysms in the temporal artery. In occluding large arteries, as after an amputation, it is always well to apply two ligatures about a half inch apart. The proximal one takes up the impulse of the heart-beat and permits tissue within the grasp of the distal ligature to heal at rest. If it is desired to occlude a vessel supplying an aneurysm, ligation in continuity is often unsatisfactory. A better plan is to apply three ligatures and divide the vessel between the distal ligatures. In this way the artery retracts, the intima is exposed to the surrounding raw tissue, secures an additional amount of nutrition from this tissue, and seems to make a firm, permanent closure, just as the endothelium does in the peritoneal cavity when exposed to a raw surface. By this procedure a permanent and firm closure is obtained, whereas a ligation in continuity that does not expose the endothelium of the intima is not infrequently followed by a reopening of the arterial channel.

Dr. Lyle (in closing):

In reply to Dr. Winslow's comment, the reason for advising excision of the aneurysm is that a study of the literature shows that ligation of the ulna and radial, or the simultaneous ligation of both, has failed in a very large percentage of the cases; failures have also occurred even after the ligation of the brachial artery.

Dr. Gibbon (in closing):

The friability of the tissues has been one of the things that has bothered every surgeon who has attempted endoaneurysmorrhaphy. I have come to the conclusion that this is partly due to the fact that the needle is not carried deep enough into the tissues, and also that the friability will not be so troublesome if catgut is used instead of silk or linen thread.

Non-pulsation is not an unusual thing in aneurysms, and, as an illustration of this statement, I would refer to the notes of the case of aneurysm of the splenic artery included in my paper.

One of the most interesting points in regard to cirsoid aneurysm Dr. Cushing has mentioned, namely, that the majority of these cases have their origin in a simple nevus, which could have been cured by excision or destruction in its early stages. One of the worst cases of cirsoid aneurysm I ever had involved the whole side of the head and face, and had existed for over forty years. It began as a small angioma of the eyelid, which had been subjected to various ineffectual forms of treatment. The patient finally had to lose her eye and was obliged to submit to repeated operations for hemorrhage, and ultimately died from this complication.

Dr. Matas (in closing):

I have the pleasure of contributing to the discussion of Dr. Lyle's paper by reporting 3 cases of restorative endoaneurysmorrhaphy operated in our clinics for aneurysms of the hand and foot. As a general rule, aneurysms of the hand and foot are treated by extirpation, but these three operations illustrate the advantages of eclectic practice in adapting methods to special indications and conditions.

Case I.—*Aneurysm of the Hand.* Radial, in the first interosseous space. The first case is that of N. S., a white farmer, resident of one of the Acadian parishes of Louisiana, aged seventy-six years. He had been gored by a cow in the first interosseous space, between the thumb and index on the dorsal aspect of the left hand. The aneurysm had been allowed to develop for nineteen years without interference,

as it caused no pain or serious disability until quite recently, when it grew rapidly and threatened to rupture. The tumor was pulsating vigorously at the time—it had attained then the size of a small potato —and the skin over it had become thin and glossy, like that of a ripe tomato. The operation was performed under local anesthesia by Dr. R. L. Stone, with the participation of Dr. Matas, at the Touro Infirmary, on June 16, 1917. A large, fairly organized clot filled the sac, which on removal revealed a single orifice of communication with the radial artery, where it enters the first interspace to form the palmar arch. A probe passed into this orifice showed that the artery was pervious, and it was decided to do a restorative endoaneurysmorrhaphy. The orifice was easily closed with fine silk without obliterating the radial, and the sac was also obliterated by a few continued catgut sutures. The patient made a rapid and uneventful recovery. (Photographs of the hand, with drawings, exhibit of the aneurysm and the operation in all its details were shown.)

Case II.—The second case is that of a negro laborer, Pete McK., aged about fifty-two years or a little over. He had been injured in the right hand by the handle of a shovel, while unloading coal. He consulted Dr. Muir Bradburn, of the Touro Infirmary staff, five months after the injury, for a pulsating aneurysm which filled the hypothenar or ulnar half of the palm of the hand. The tumor projected prominently as a conspicuous eminence over the ulnar border of the hand. It collapsed on pressure, and the pulsations were arrested by compression of the radial and ulnar arteries and refilled promptly on release of pressure. The patient was markedly arteriosclerotic and gave a Wassermann positive reaction. Under local and regional anesthesia, on June 7, 1919, Dr. Bradburn opened the sac and, after removal of the clot, exposed two slit-like orifices which communicated presumably with the ulnar artery. The orifices were three-eighths inch apart and were closed separately with fine silk. The sac was then obliterated by catgut sutures and the skin incision closed with interrupted silkworm. The sutures were removed on June 14. On June 21 the patient was hoeing, and on July 21 reported that he had been chopping wood for a week. When seen again, November 19, the hand was perfectly normal in appearance and function. (Photographs showing the tumor before and after the operation were exhibited through the courtesy of Dr. Bradburn.)

Case III.—*Aneurysm of the Dosalis Pedis Artery.* The third case is of aneurysm of the left dorsalis pedis. The patient, Sidney G., a farmer, aged thirty-seven years, had noticed a small pulsating tumor in the

dorsum of the foot for the last twelve years. The tumor had appeared apparently without cause, and as it remained indolent and caused no pain and inconvenience he had not worried about it. Two weeks before admission a block of wood fell on the back of the foot and struck this same little spot. He suffered no immediate disagreeable consequences from the injury, but shortly afterward (a day or two) he noticed that the little tumor was growing larger and becoming painful, so much so that it interfered with his work. When first seen at the clinic (Charity Hospital) the tumor was about the size of a small pullet egg, lying right under the skin, pulsating vigorously with a characteristic expansile pulsation. On April 12, 1923, Dr. Matas, under local anesthesia, opened the sac and extracted a firm, dense, partially laminated clot which had molded itself to the shape of the sac. After removal of the clot an oval opening, about a half inch in length, was exposed in the posterior and outer wall of the sac, near its upper pole. On exploring this opening with a probe it was found to lead into a channel which communicated above with the dorsalis pedis and below with another vessel, presumably the anastomotic branch of the external plantar. This channel no doubt represented the primary dilatation of the artery, which had probably been ruptured by the blow of the block of wood two weeks before. The sac wall was smooth and lined everywhere with endothelia. A continuous fine silk suture carried on an ophthalmic needle was now made to close the opening completely. The collapsed walls of the sac were brought together and enfolded with catgut, and again brought in apposition with through-and-through silkworm sutures, tied on each side of the incision over rolls of gauze. The edges of the wound forming an elevated cutaneous crest were sutured with continued catgut. The patient made a perfect recovery and was discharged well in three weeks.

Conclusion. This case shows, as in the two preceding instances (hand aneurysms), that it is not only practicable to do a restorative operation in an aneurysm of a small vessel at the extreme periphery of the body, but that this procedure is technically easier than extirpation and certainly less traumatizing.

PLEXIFORM NEUROFIBROMA OF THE SOLAR PLEXUS

By HERBERT A. BRUCE, M.B., L.R.C.P., F.R.C.S. (Eng.)
TORONTO, CANADA

One of the most valuable contributions to the subject of tumors of nerves was that made by Mr. Alexis Thomson, late Professor of Surgery in the University of Edinburgh, in a monograph on *Neuroma and Neurofibromatosis*, published in 1900. All present will have a pleasant memory of the charming personality of Professor Alexis Thomson, who was the guest of this Association in Washington in 1913, when he gave us an admirable paper on "Fibromatosis of the Stomach and its Relationships to Ulcer and to Cancer." I am sure the Fellows will be deeply grieved to learn of his sudden death last month, at Gibraltar, while returning home from a three months' holiday in the East.

The term "neuroma" was introduced by Odier, of Geneva, in 1803, to designate "tumors formed by diseased enlargements of the nerves." William Wood, of Edinburgh, discussing the origin of neuromata in a paper in the *Transactions of the Medico-Chirurgical Society of Edinburgh*, in 1829, believed them to originate from the connective-tissue sheaths of the nerve and not from the nerve tissue itself. He remarked upon the inefficacy of medicines and recommended removal of the tumor by operation. Among others he quoted a case from the *Encyclopedie Methodique de Chirurgie* (Paris, 1792), in which amputation was performed for what was undoubtedly a plexiform neurofibroma of the median nerve of the forearm. Smith, of Dublin, in his classical work on *Neuromata*, in 1849, considered that the mere anatomical connection of a tumor with a nerve was sufficient to regard it as a neuroma, whatever its structure might be. How-

ever, Virchow, in his lectures, published in 1863, placed the pathology of neuromata on a new basis by classifying neuromata on a structural instead of a clinical basis and dividing them into the true and the false, the former composed mainly of new nerve tissue, the latter (pseudoneuroma) of connective tissue derived from the sheaths of the nerve. True neuromata are very rare, but several authentic cases have been recorded.

Attention is called to the present case of plexiform neurofibroma of the solar plexus, not only because of its inherent interest and rarity, but because it has to be taken into consideration in a differential diagnosis of abdominal tumors. However, as I am only able to find one other case of plexiform neurofibroma affecting the solar plexus in the literature, which case is given by Bruns in his paper in the *Beitrage zur klinischen Chirurgie*, Bd. viii, it will not be necessary to work out any elaborate methods for its detection.

Mr. J. Kingston, aged thirty-five years, referred to me by Dr. R. J. McMillan, gave a history of having been injured in a fall at the age of eighteen. He had never had any serious illness until an attack of influenza four years ago, which confined him to bed for three months. Since that time he has had constant pain in his back in the right lumbar region, accompanied by tenderness on deep pressure, usually relieved by a belladonna plaster. At irregular intervals, increasing of late, this pain in his back would be so severe as to confine him to bed for several days, and was generally regarded as lumbago. When free from these attacks he had a good appetite, appeared to be in good health, although always very thin, and carried on his usual work. The tumor became palpable about three months before I saw him. Two months ago he suffered from lienteric diarrhea, which cleared up on milk diet. For the last few weeks he has not been able to retain any solid food, and has even vomited most of the fluids given. When he entered the hospital his urine was found to contain diastase. Examination of the abdomen disclosed a tumor, about the size of a grapefruit, in the midepigastric region,

FIG. 1.—About one-tenth of tumor mass; nodules are approximately the actual size. A, dissected portion of tumor; B, dissected portion of tumor section; C, undissected portion of tumor.

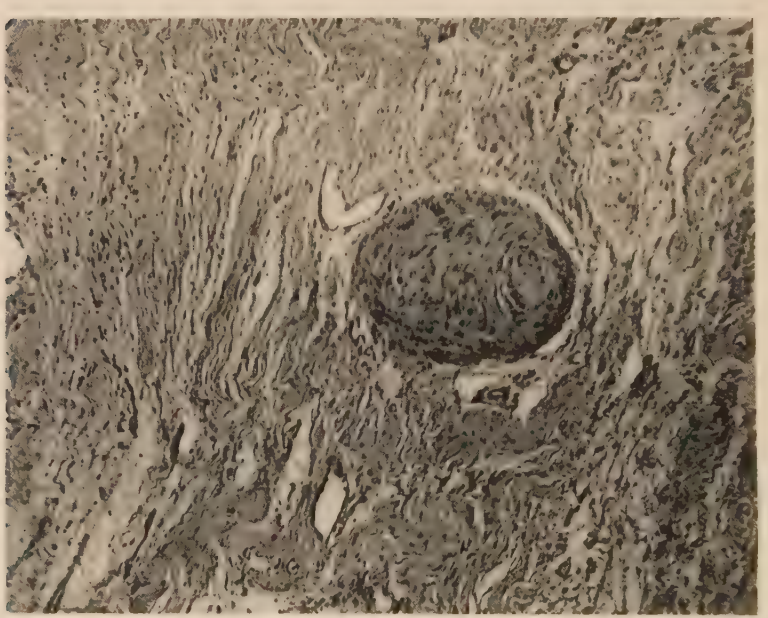

FIG. 2.—A non-medullated nerve fiber surrounded by well-formed connective tissue. · Low power (× 5 eyepiece).

only very slightly movable from side to side. It felt solid and had a tympanitic note over it. The abdomen otherwise was very thin, tense and emaciated. There was no pigmentation of the skin, and there were no nodules seen in any other part of the body.

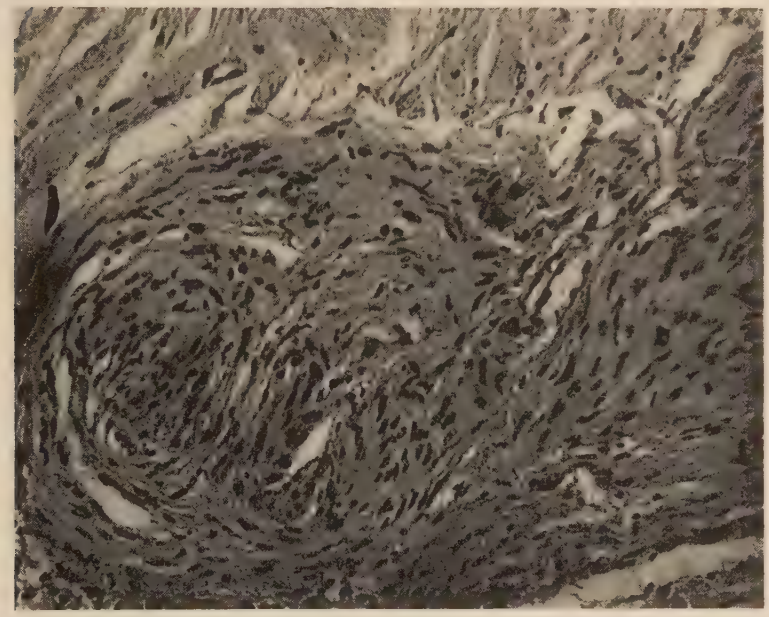

FIG. 3.—Whorl of non-medullated nerve fibers with the surrounding loose edematous connective tissue. Low power (× 10 eyepiece).

An *x*-ray examination was made by Dr. H. M. Tovell, who reported as follows:

"There is no definite radiographic evidence of a pathological condition in the stomach or bowel which would indicate ulceration or malignancy. There is evidence that a mass external and posterior to the stomach is pressing upon the lesser curvature and also upon the transverse colon. From the position of the mass one would be suspicious of the pancreas, yet there is no evidence of displacement of the duodenum, which assumes the

usual relation to the stomach, and which one would think would not be the case if a pancreatic tumor were present."

However, a diagnosis of "tumor of the pancreas" (probably a cyst) was made, and it was thought that the injury referred to above might have been a causative factor. Under anesthesia the tumor, which was very hard and nodular, could be moved freely from side to side, and to a less extent from above downward. On making the incision the transversalis fascia was found tightly

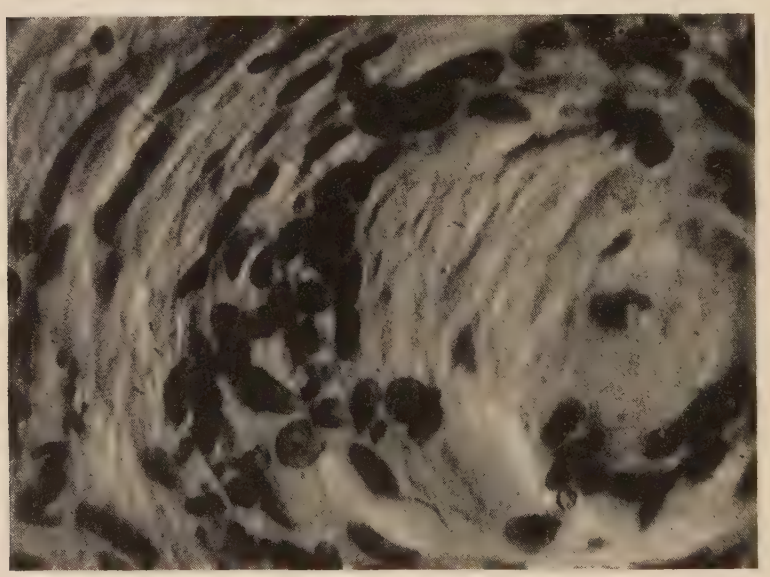

FIG. 4.—Cells in the whorl formation. High dry power (× 10 eyepiece).

stretched over the tumor, and when it and the peritoneum were divided the tumor popped out as if under great pressure. It was then seen to press forward the gastrohepatic omentum and the stomach, which was tightly stretched over it. The pylorus, which was lying in the middle line, was widely dilated, as were also the first and second portions of the duodenum. The glands along the greater and lesser curvatures were not involved. The tumor mass was about the size of a grapefruit, distinctly nodular, and

appeared to be connected with the pancreas. Thinking it malignant and inoperable, after removing a couple of nodules for microscopical examination, the abdomen was closed, but with considerable difficulty.

We were surprised at receiving a report from the pathologist that the section was a myoma. In view of these findings, and the fact that he was unable to retain any nourishment, owing to the mechanical effect of the tumor in shutting off the outlet of the stomach, making a fatal result inevitable in a very short time, we decided to try and remove the tumor. The former incision was reopened twelve days later and a thorough examination of the tumor, both through the gastrohepatic omentum and the transverse mesocolon, revealed it lying behind the pancreas, which was not involved. A good deal of difficulty was met with in dissecting out the growth, as a number of very large blood-vessels coursed through it. The mass looked a little like a thick tapioca pudding, with a fine mesh of fibrous-tissue stroma between the little nodules. The patient recovered nicely from the immediate effects of the operation, and the same evening his pulse was good and his condition seemed satisfactory. However, he died suddenly the next morning, probably from pulmonary embolism.

I am indebted to Dr. G. W. Lougheed, M.C., pathologist to the Wellesley Hospital, for the careful dissection and examination of the tumor, and the following report:

"*Gross Specimen.* Consists of a large tumor mass which is growing in an irregular fashion. There are a large number of grape-like masses projecting from the inner surface of this tumor. They are firm in consistency and whitish in color. On section they have a slight tinge of green. The remainder of the tumor is red in color and appears to be covered with a firm, thin sheet of fibrous tissue. Beneath this are more of these grape-like masses.

"*Microscopical Findings.* Slides of some of these nodules show them to be made up of a loose areolar connective tissue with numerous nerve bundles running through it. There is a fair amount of collagen material present, and in places the tissue is

rather edematous in character. There is no evidence of ganglion nerve cells present. The majority of the tumor is made up of non-medullated nerve fibers intimately surrounded by fibrous tissue.

"*Diagnosis.* Neurofibroma (plexiform)."

The plexiform is the most interesting type of neurofibromatosis. It is a fibromatosis confined to one or more contiguous nerves or a plexus of nerves. The lesion is the same as the more generalized form of neurofibromatosis, and the two conditions may be met with in the same individual. Although rare, Bruns collected 42 cases and Thomson collected 18 cases, which with 2 of his own make a total of 62 cases. It is stated that the tumor may undergo degeneration, and may even disappear (Baumgarten), but Thomson agrees with Goldmann that such changes are altogether exceptional. A peculiarity of the plexiform type is the tendency, although involving a purely sensory nerve, to penetrate the adjacent muscles. There may be an overgrowth of subcutaneous tissue and skin in relation to a plexiform tumor, which is best described by the term "elephantiasis," where the skin frequently shows excess of the normal pigmentation. By far the most frequent situation of plexiform neurofibromatosis is the subcutaneous tissue of the head and neck—in the distribution of the trigeminal and superficial cervical nerves, although it may be met with anywhere throughout the sympathetic and peripheral cerebrospinal systems.

The following table is given by Bruns with additions collected by Thomson:

SITES OF PLEXIFORM NEUROFIBROMA

	Cases.
Temple, forehead and upper eyelid	18
Posterior part of neck and behind auricle	14
Nose and cheek	4
Region of lower jaw and anterior half of neck	5
Breast and back	8
Extremities	9

The nerves of the extremities include the median, musculospiral, radial and small sciatic. Goldmann and Pomorski have

each recorded a case in which the intercostal nerves were affected, the tumor projecting into the pleural cavity. Bocasso observed a tumor below the jaw, passing along the hypoglossal nerve into the substance of the tongue. Bruns drew attention to the fact that the most common seats of the plexiform neurofibroma are also the most common situations of elephantiasis neuromatosa or pachydermatocele, with which it is frequently associated.

Plexiform tumors are found coexisting with a generalized fibromatosis, and with multiple skin fibromata (molluscum) in about 50 per cent of cases.

Plexiform neurofibroma resembles the elephantiasis with which it is commonly associated, inasmuch as it may be present at birth or develop in early infancy. As to the influence of heredity, a study of the cases demonstrates the fact that neurofibromatosis is sometimes inherited and sometimes met with as a family disease, and gives support to the view that the chief etiological factor in the disease is one which dates from intrauterine life. Other defects of developmental origin are frequently seen associated with this disease. So far as our present knowledge goes, Bruns, Goldmann, Thomson and others consider that the condition is a form of giantism or elephantiasis of the connective-tissue elements of the peripheral nervous system.

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NEUROEPITHELIOMA OF THE SPINAL CORD: A CLINICAL AND PATHOLOGICAL STUDY

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NEW YORK

AND

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NEW YORK

The opportunities for the study of tumors of the central nervous system and its meninges are becoming so plentiful that rapid progress in this field may confidently be expected. The early statistics collected by Bruns,[1] Schlessinger[2] and others related to neoplastic growths of all types, and only in recent years has it become recognized that further advance will be made most surely if the study of individual types be made. When, therefore, one observes a tumor of the spinal cord which presents unusual features, the report of such a tumor as an isolated experience is more than justified. For this reason we venture to tax your attention with considerable detail.

In attempting such a report one encounters difficulty in determining to what extent the tumor under consideration differs from those described by other observers. The nomenclature used in the literature is confused, and in many instances, particularly of clinical reports, the name applied to the tumor is not sufficiently distinctive, and no histological data are given on which to base accurate information.

The tumor used as the text of this discussion was entirely extramedullary. So far as can be determined it took its origin from beaneath the pia-arachnoid, the latter membrane passing over it and binding it closely to the cord. It differed from most extramedullary spinal tumors in that it possessed excessive malignancy.

Joppich,[3] in 1903, found only 24 cases of extramedullary leptomeningeal sarcoma recorded in the literature. Schlessinger,[2] in analyzing 224 autopsy findings of cord tumors, includes 107 sarcomas. In many of these reported cases the striking characteristic of extramedullary tumors, even when called sarcoma, is their relative non-malignancy. Hunt and Woolsey[4] report an extradural fibrosarcoma which recurred in fifteen months after removal. A second operation was thought to have incompletely removed the growth, and yet recovery took place and the patient reported entirely well four years later. Inglis, Klingman and Ballin[5] report one extramedullary glioma. It was attached to the subpial neuroglia of the cord and the posterior root of the 7th thoracic nerve. Operative removal was successful and the patient was well eighteen months later.

Malignant tumors are of common occurrence in the substance of the spinal cord, but in most instances they are definitely infiltrative of the cord, which is in marked contrast to the one discussed here. We have designated this tumor as a neuroepithelioma. Schlapp[6] reports a spinal tumor under this name which occurred as an isolated growth in the dorsolumbar region of a cord which was the seat of a central gliosis. In this respect it paralleled our tumor. In the literature on the cases of neuroepithelioma of the cord he finds that all of the authentic and well-examined cases, with the exception of one or two, are accompanied by a central gliosis. In these exceptions the tumor had destroyed so much of the cord that it would be impossible to say whether there was or was not an underlying gliosis present. Schlapp concludes that a central gliosis is a condition almost constantly found in cases of neuroepithelioma, and he advances the view that the former stands in a causative relation to the latter. In our case the gliosis seems of a later date than the tumor.

The patient in whom the tumor developed was a woman in her fortieth year, who first came under observation on March 20, 1922. She had been married twice and was the mother of three

children, the last born in June, 1921. There was no suspicion of tuberculosis or lues. Her previous history, except for one incident, was entirely irrelevant. In November, 1919, she was thrown from her horse while hunting, and suffered a "twist of the neck." This caused little immediate inconvenience, but

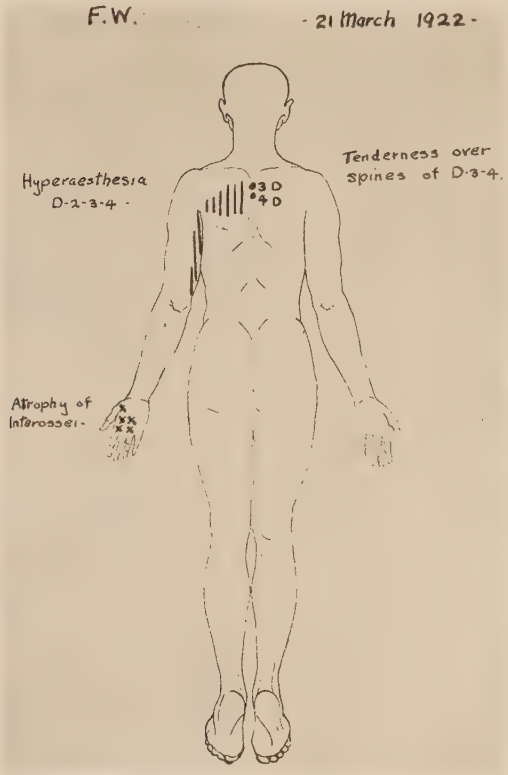

FIG. 1.—Chart showing the objective findings on March 21, 1922. Hyperesthesia is represented by dark vertical lines.

three days later the left shoulder and arm pained her rather severely and became stiff. No evidence of any nerve pain at this time was obtained. She was treated by manipulation, and after a few treatments all symptoms subsided. No further pain was experienced until July, 1921, twenty months after the

injury, when about six weeks postpartum there was a return of the same pain, and in addition a pain under the left scapula. This attack was more obstinate and only yielded after several weeks of treatment with manipulation, electricity, baking and

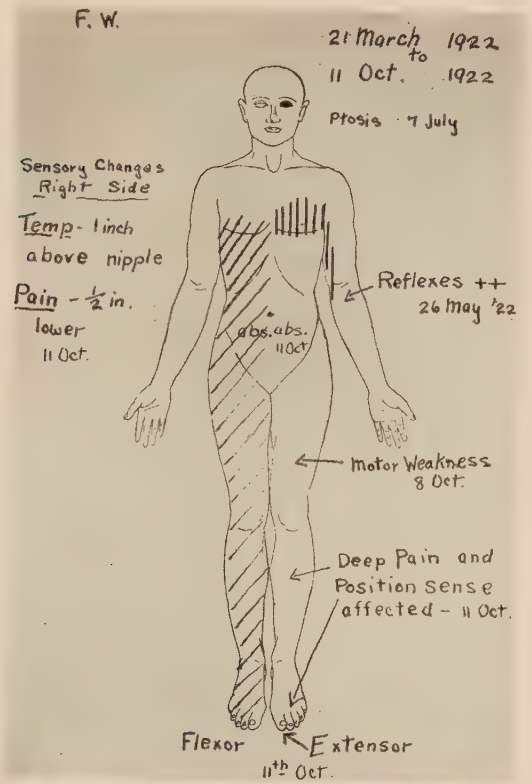

FIG. 2.—Chart showing sensory, motor, reflex, cervical sympathetic changes from March 21 to October 11, 1922. Sensory loss over right side of body indicated by diagonal lines.

finally a visit to the baths at Hot Springs. She remained entirely free from pain until January, 1922. At first the pain was not severe, but in March there was a sharp recurrence, from which she never recovered. On one occasion, some time in February, before the pain became severe, she noted an involuntary flexion

of the middle finger of the left hand. This had not recurred and there had never been any other muscular involvement. From the onset of this attack in March the pain had been unbearable, and 1 or 2 grains of codeine were taken every night to permit of any sleep. The patient was an unusually

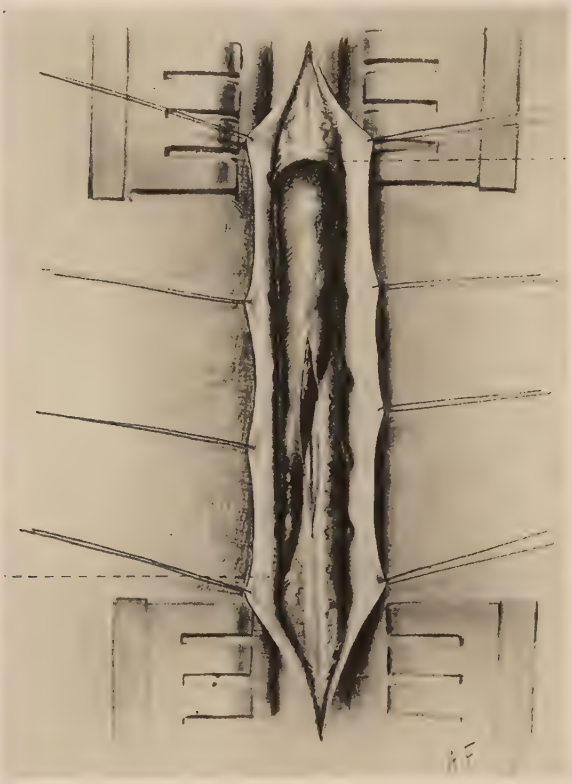

FIG. 3.—Drawing to show the original tumor as exposed at operation covered by the arachnoid membrane.

healthy appearing woman who gave evidence of being in severe pain. The usual physical examination failed to disclose any defects not connected with the nerve lesion except the presence of a root abscess on one of the upper molar teeth. The presence of this had been known a year previously, when it was demon-

strated with the radiograph, but it had received no treatment. A neurological examination was recorded in the following note: There is excruciating pain in the entire left upper extremity, including the upper scapular region. This entails some involvement of all the nerves from the 4th cervical to the 3d dorsal.

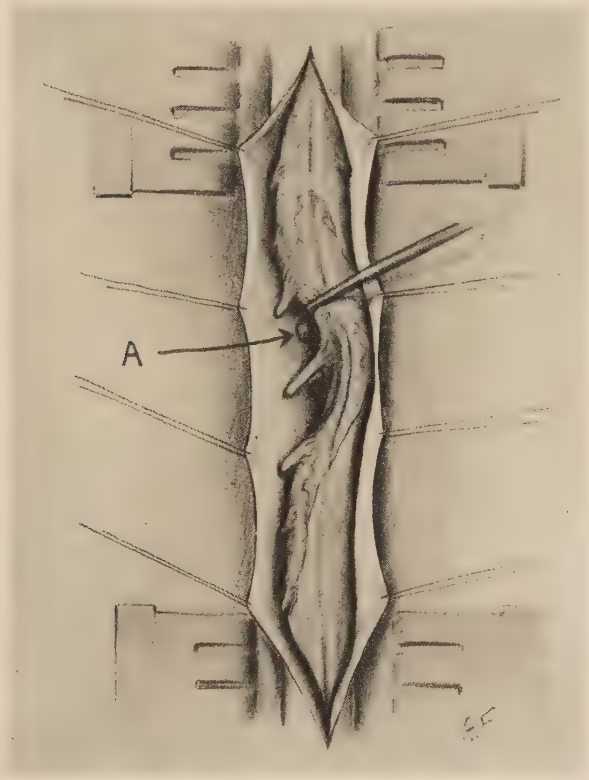

FIG. 4.—Drawing to show a secondary tumor nodule lying in the dura mater as exposed at operation. A, Secondary nodule in dura.

She describes this pain as tearing, burning and boring. It is made markedly worse by any movement, but becomes unbearable unless the position of the arm is changed from time to time. There is also hypersensitiveness in the arm, forearm and hand, most marked over the distribution from the 1st and 2d dorsal

segments. There is a definite zone of hyperesthesia anteriorly and posteriorly along the left 2d and 3d costal spaces, showing involvement of the corresponding nerves of the 2d and 3d dorsal segments. The cervical and brachial nerve trunks are tender, and light palpation along their course increases the pain in the

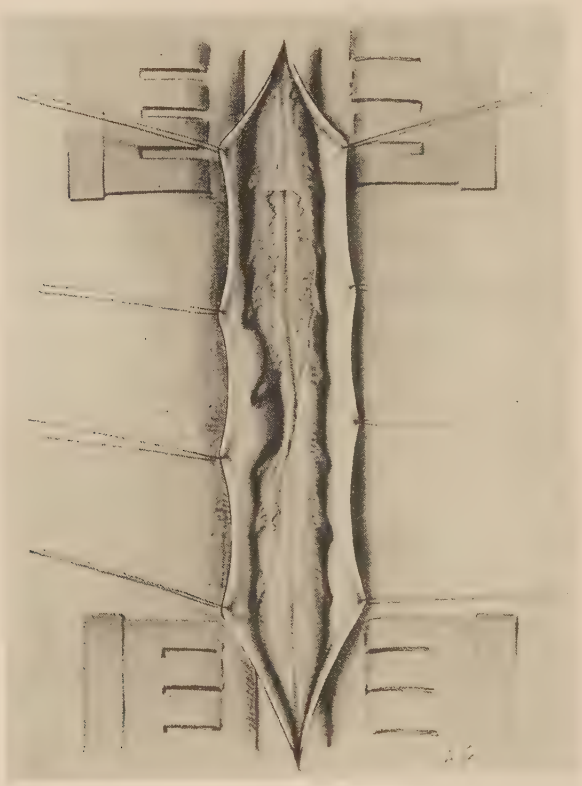

FIG. 5.—Drawing to show appearance of the cord after operation.

scapular and arm regions. The third dorsal spine is tender on percussion, as are the 1st and 2d to a less degree. Flexion and extension of the neck increase the pain. There is atrophy in the first interosseous space of the left hand, and the whole hand seems smaller than the right. The skin is shiny. There is no

FIG. 6.—Low-power view of section from the original tumor removed at operation.

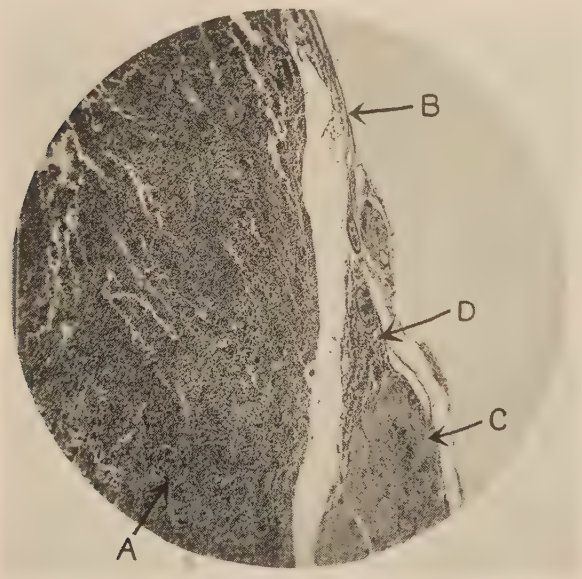

FIG. 7—Low-power view of tumor removed from anterolateral aspect of cord at autopsy. A, Tumor composed largely of cells (neuroepithelioma); B, arachnoid membrane covering tumor and nerve root; C, an anterior nerve root stretched over the tumor; D, tumor cells invading sheath of nerve root.

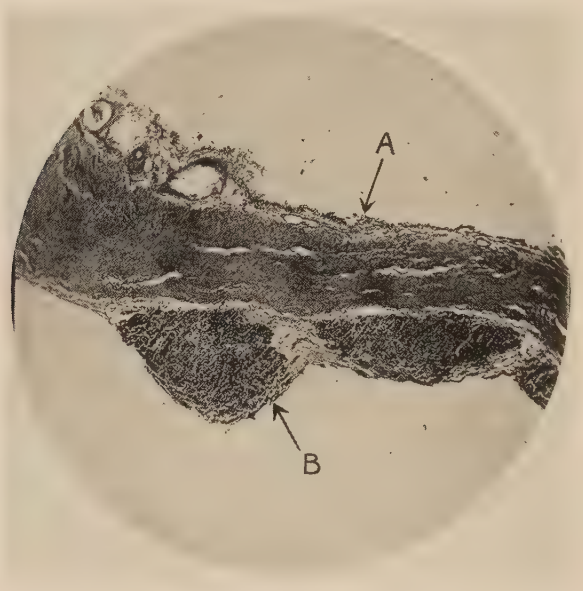

FIG. 8.—Metastatic nodule on under surface of the dura mater near the original tumor. A, Outer surface of dura mater; B, metastatic tumor nodule.

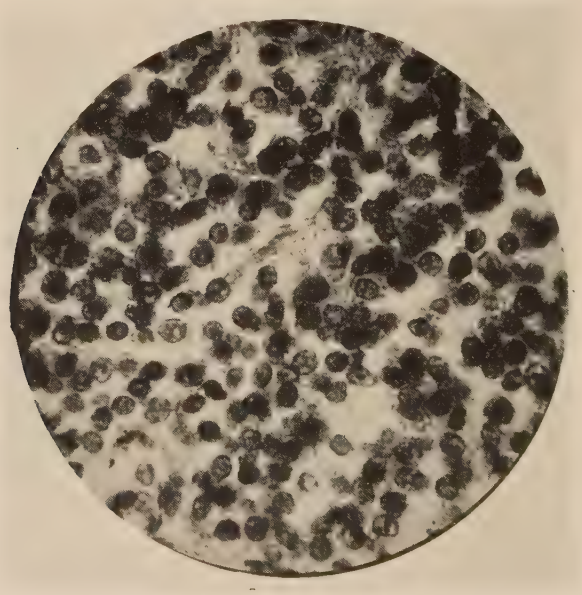

FIG. 9.—High-power view of tumor cells to show the neuroepithelial cells with intercellular fibrils.

paresis or muscular weakness other than that due to pain on using the muscles. There are no changes in any reflexes of the trunk or lower extremities. Those of the left arm and hand seem normal, though pain disturbs the examination. An *x*-ray examination of the molar tooth confirmed the presence of the abscess, and two days later the tooth was extracted and the

FIG. 10.—Section of the spinal cord above the level of the tumor to show pencil of gliosis within the cord. A, Area of gliosis.

abscess cavity cleaned. An *x*-ray examination of the spine and upper extremities revealed no abnormalities, and no cervical rib was present. A tentative diagnosis of a cervicodorsal radiculitis was made, with the tooth abscess as an etiological factor, though the fear of a neoplasm of the cord or spine was entered in the notes on that occasion.

On March 22 Dr. Frederick Tilney saw the patient in consulta-

tion, when the following note appears in the record: "Dr. Tilney confirms the findings as given and concurs in the interpretation of neuritis or radiculitis, with, however, the possibility existing of vertebral or medullary lesion," probably neoplastic(?). Dr. Tilney emphasized the fact that the hyperesthesia extended to the distribution of the posterior dermatonic divisions of the

FIG. 11.—Section of the spinal cord below the level of the tumor showing the pencil of gliosis within the posterior horn of the cord. A, Gliosis; B, zone of hemorrhage about the gliosis.

lower cervical nerves. Such findings suggested an intravertebral lesion.

The usual treatments were instituted, and on March 31 the following note was made: "The patient seems to have improved in general condition and the pain is slightly less some of the time. Treatment has been absolute rest, with application of

heat, anodynes being given sparingly. Seen with Dr. Foster Kennedy, who confirms all the findings, and believes the condition is a pachymeningitis around the foramina for both the cervical and dorsal nerve roots. He thinks this would account for all symptoms, the process being more extensive than usual. He finds nothing on which to base the diagnosis of a bony or

FIG. 12.—Section of the cord lower down than in Fig. 11 to show an additional area of gliosis within the white substance of the posterior column of the cord at "A."

medullary neoplasm at this time. Advises addition of salicylates to the treatment."

The course of the condition continued unchanged for the following six weeks. There had been no real cessation of pain, and it had been necessary to give anodynes rather freely. Repeated examinations failed to show any symptoms which would implicate

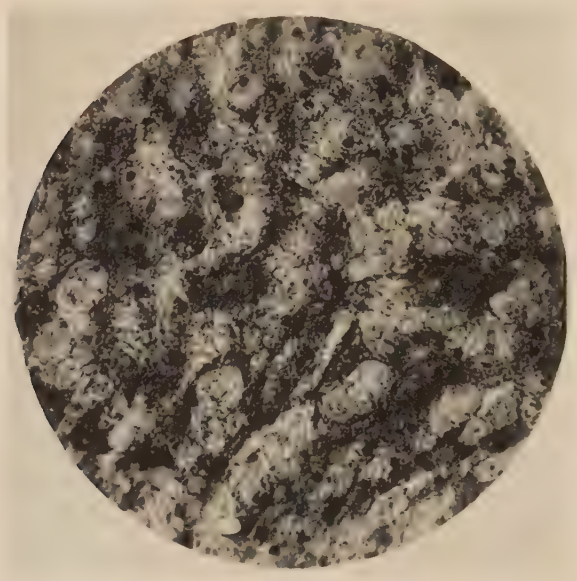

FIG. 13.—High-power view of gliosis seen in Figs. 10 and 11.

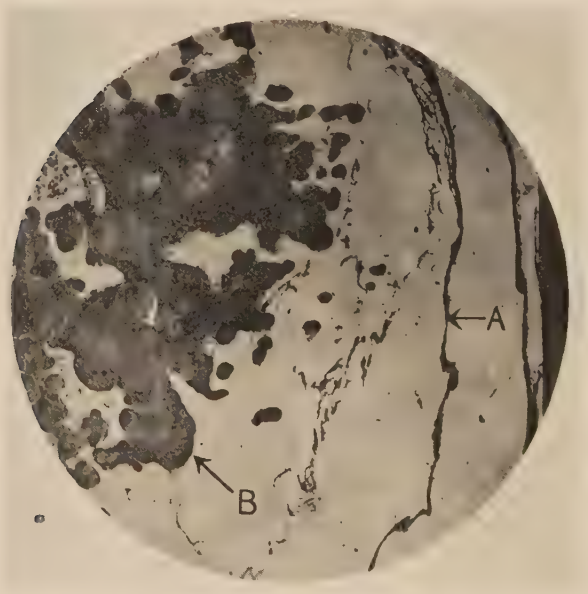

FIG. 14.—Section of one of the small bony plaques found in the arachnoid below the level of the tumor. A, Arachoid; B, bony plaque.

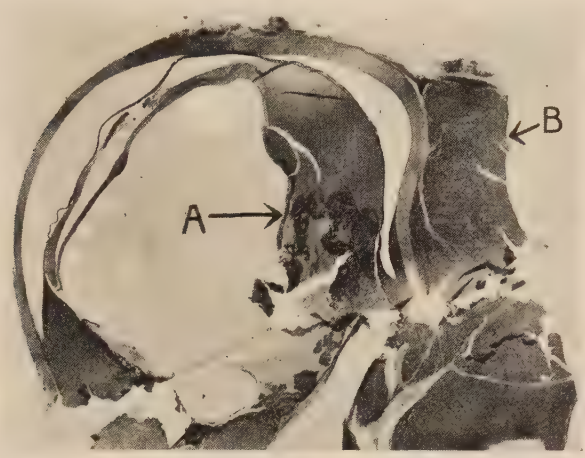

FIG. 15.—Section of an eye affected by neuroepithelioma of the retina (so-called "glioma retinæ"). A, Intrabulbar portion of the tumor which shows typical rosette formation when viewed under high power; B, extrabulbar portion of tumor which is devoid of rosettes and resembles the tumor described in this paper. (Section loaned by Doctor Samuels.)

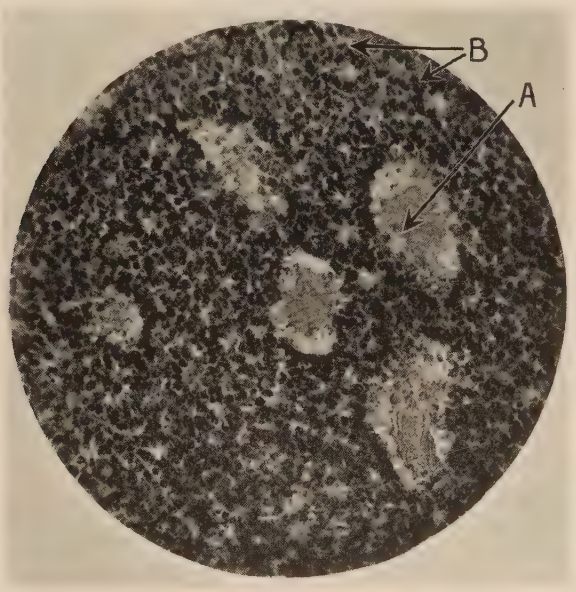

FIG. 16.—High-power view of intrabulbar portion of tumor shown in Fig. 15. A, Typical "rosette" formation of neuroepithelioma. B, Secondary or smaller rosettes so often seen in neuroepithelioma of the retina.

the cord itself in the picture. The pain had been so excruciating that the patient's general condition was suffering, though no more than is often seen with a severe cervical radiculitis.

On May 26 Dr. Kennedy again saw the patient. He could find no new manifestation, except the sympathetic involvement, mentioned below, and an increase in the left arm reflexes, and adheres to the diagnosis of posterior root pachymeningitis, with

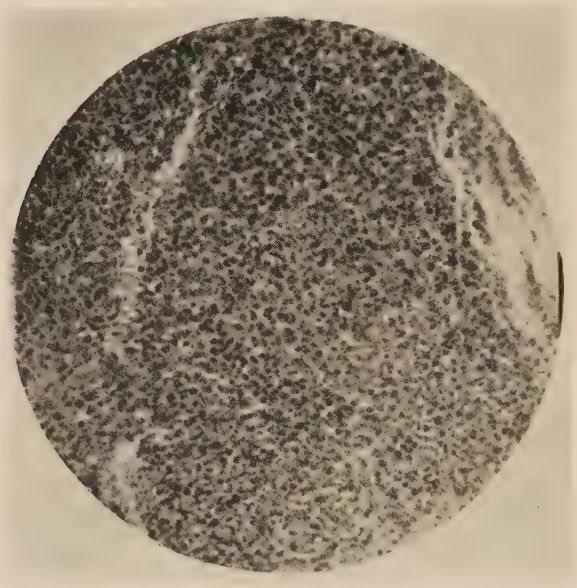

FIG. 17.—Section of the extrabulbar portion of tumor shown in Fig. 15 showing absence of rosette formation. The cells of the retinal neuroepithelioma are smaller than those of the spinal cord and adrenal tumors.

pressure. He thinks there is only a minimal involvement of the anterior roots. In general, he finds an improvement since the last visit. The reflexes, with the exception named above, are all normal, and it is particularly noted that the abdominal reflexes are equal and brisk. The hyperesthesia seems less severe, but the zone on the chest is more extensive now, involving the 8th cervical and 1st dorsal segments. The atrophy has not

increased. There are no anesthesias or pareses demonstrated. An additional symptom was noted at this time—the nurse first observed it a day or two previous to this visit—in the presence of a false ptosis of the left eyelid and inequality of the pupils, the left being smaller than the right, an involvement of sympathetic fibers. Later marked unilateral sweating of the face was present. We continued in the belief that we were dealing with

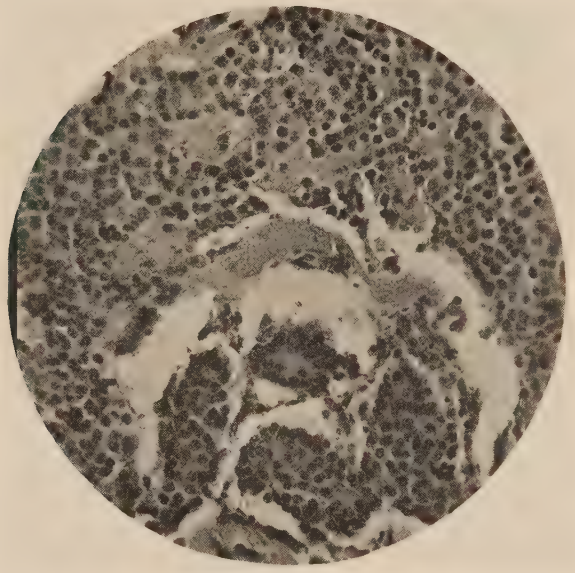

FIG. 18.—Section of a neuroepithelioma of the adrenal showing rosette formation and also formation of neurofibrils without typical rosettes. (Dr. James Ewing's Section.)

an inflammatory lesion, which possibly had its cause in the tooth abscess, though the correction of this had disappointed our hope that a cure might follow. An examination of the stools showed an excessive fermentation, with heavy content of Streptococcus viridans, and it was considered possible that the irritation was kept up from this source.

Repeated examinations failed to add a single finding which

would confirm the suspicion constantly in our minds that a cord neoplasm was present. There was nothing in the picture which was incompatible with a simple cervicodorsal radiculitis. The very intensity of the pain, its constancy and its type were all in accord with this conception.

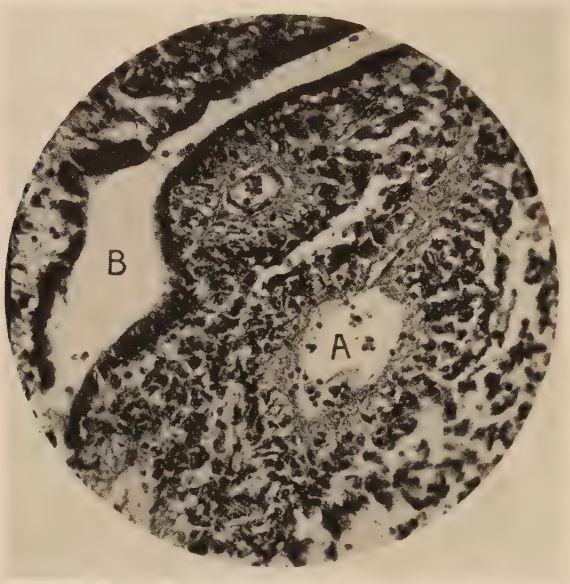

FIG. 19.—Section of a non-malignant neuroepithelioma of the spinal cord removed after a clinical course of seven years. This tumor shows the much less cellular type of tumor with very typical rosette formation and, in addition, a cavity lined by neuroepithelium. A, Rosette with abundant neurofibril formation; B, cavity lined by neuroepithelium.

July 5: The progress has been slowly toward recovery. The anodyne (morphine mostly), which had been required in increasing amounts, was withdrawn during the middle of June. Since that time the patient has been without anything to relieve pain other than an occasional dose of luminal or trional to induce sleep. The pain had grown intermittent so that sometimes she would be free for several hours, and on occasion would sleep all night without medication. At times, however, she had very

severe burning sensations around the elbow on the inside of the arm (1st dorsal segment) and sometimes in the hand. There were also clonic contractions of the arm muscles, mostly the anterior group, which were painful. These could be controlled by mental influence and reassurance, and were believed to be more due to a lowering of her morale from the long suffering than from an anterior-root irritation. Voluntary use of the extremity had improved and the muscular atrophy seemed decreasing.

The streptococci in the intestine were still abundant, and for this she had been receiving an acid bacillus tablet with milk sugar and an autogenous vaccine.

During the summer months there seemed to be a slow but steady improvement. The atrophy of the hand and arm muscles became less marked, and the intense pain was absent for considerable periods. The patient was up for a time each day, and sleep was obtained with small amounts of allonal. Repeated examinations failed to show any changes other than those recorded at the first examination. No further alteration in any reflexes was observed. There was no loss of any form of sensation in any part of the body. No paralysis or paresis could be demonstrated. The painful clonic contractions of the upper extremity had stopped. There was still hyperesthesia and hyperalgesia in the region of the left scapula, left arm, forearm and hand, and the region of the left third costal space anteriorly and posteriorly. The improvement seems to be coincident with the freeing of the intestine from the streptococcus and thereby lent support to the belief that we were dealing with a toxic cervicobrachial radiculitis.

About the middle of September fears of a neoplasm were again aroused by the appearance of a pain beneath the right scapula and a tingling sensation in the right hand. These were transitory and not very definitely localized by the patient, but at times were quite severe. On one occasion the nurse reported that the patient had commented on some peculiar sensation in the skin of the right thigh and groin, a passing illy-defined phenomenon. This was the first evidence that any cord disturb-

ance might be present, but a thorough examination failed to reveal any finding which would confirm this.

October 7: The pain in the right upper extremity has increased and at times has been very severe in the neighborhood of the scapula. There has been no abnormal sensation in the hand. The left upper extremity seems to be constantly improving. The pain is definitely lessening. Atrophy is decreasing and the ability to move the arm without undue pain is greater. There is added today a new symptom, namely, a weakness in the left lower extremity when walking, so that some part of the extremity—the patient thinks near the hip-joint—seems to give way. This is not present at all times. There is also described in the right lower extremity various paresthesias, "as though asleep, and there is a creeping sensation as though I can feel the blood circulating under the skin." There is no actual pain and no pin-and-needle sensation. These findings make probable a cord compression.

October 11: Neurological examination made today gives the following findings: In the upper extremity there is a continued improvement on the left side in every respect. No changes are found in the right extremity. The reflexes are present and are apparently equal on both sides. The same tenderness, though less marked, as originally existed in the spinous processes of the upper dorsal vertebræ, is present. No fibrillary muscular contractions are present. The abdominal reflexes are altered on both sides: On very excessive stimulation there is a response on the left side, but none on the right. The knee reflexes are slight exaggerated on both sides and ankle-clonus is present on both sides. There is a doubtful Babinski present in the left great toe. The eye condition is still present, though less marked than formerly.

Sensation. Below the level of the second dorsal segment on the right side there is a definite dulness of sensation both to tactile and painful impressions. The patient says that everything feels as though the part were numb. There is a definite loss of appreciation of hot as compared with cold stimuli below

this level. There is much disturbance of deep pressure pain sense and of the sense of position in the left calf and foot, respectively. There are no sphincteric disturbances. The left lower extremity is definitely weaker than the right, but the patient can walk with no perceptible limp. All these findings seem to indicate a pressure on the left side of the cord at the 1st or 2d dorsal segment.

October 12: Seen by Dr. Van Horne Norrie in consultation. He agrees with all the findings in the examination and arrives at the diagnosis of compression of the left side of the cord, probably of tumor origin, though, of course, this is uncertain. While the center of the compression is at the 1st or 2d dorsal segment, it must also reach up to the 4th or 5th cervical in order to involve the brachial plexus, and may readily extend down to the 4th or 5th dorsal because of the root pains on the right side extending down to the 5th or 6th segment.

A spinal cord tap was done and 3 or 4 cc of an orange-tinted fluid withdrawn under low pressure. An examination of the spinal fluid was as follows: Bright yellow fluid which did not clot on standing. Clear, small amount of macroscopic blood in bottom of tube.

Cells, 5 per cm. Smear showed very few cells; all mononuclear.

Culture, sterile.

Albumin, 3 plus.

Globulin, 2 plus (Noguchi and Nonne).

Sugar, 2 plus.

Wassermann, negative.

Colloidal gold curve, negative.

October 17: From the previous note until this date, when the operation was performed, there was a steady increase in the symptoms of a cord compression. The right side below the level of the 2d dorsal segment showed a loss of sensation of all types. The left lower extremity was almost completely paralyzed and the right had grown so weak that the patient could not bear any weight upon it. All reflexes were exaggerated except the abdominals, which were completely absent, and there was a marked Babinski on the left and a doubtful one on the right.

A laminectomy was performed according to the usual method, the laminæ of the 6th and 7th cervical and four upper dorsal vertebræ being removed. The tumors as described below were removed by sharp dissection without difficulty, and with no undue trauma to the cord. The left lateral aspect of the spinal cord beneath the laminæ of the 6th and 7th cervical and 1st and 2d dorsal vertebræ was the site of a club-shaped, very friable, cellular appearing tumor. The thicker portion of the tumor was opposite the 6th and 7th cervical laminæ, and the thinner portion opposite the 1st and 2d dorsal laminæ. The tumor was a dark, purplish color and was intimately adherent under the arachnoid to the substance of the spinal cord. It had very seriously compressed the cord at the level of the 1st dorsal segment, but had not extended around the cord either anteriorly or posteriorly. The compression appeared to have been due to the tumor's position beneath the arachnoid, and not because of any lack of room in the spinal canal. Opening of the dura did not relieve the compression.

If the spinal cord and tumor had been removed intact and hardened in formalin before section the shape of the tumor would have been as follows: Beginning above, at a point beneath the interval between the 5th and 6th cervical laminæ, the tumor was spread out over the left lateral aspect of the cord and retained thus by the arachnoid. Descending downward, the superficial envelopment of the cord by the tumor laterally became rather less, but the extent of growth from left to right increased to a maximum point opposite the 1st or 2d dorsal segment in such a way as to exert more pressure on the cord at this point than it did above, where the surface expansion was greater. From this latter point downward the tumor gradually thinned out in all its diameters. From the clinical symptoms and this shape of the tumor it is fair to assume that the tumor made its first appearance close to the 1st dorsal segment, and at first grew between the arachnoid and cord as a more or less rounded mass. As resistance increased, it extended upward as a thinner, more enveloping growth between the cord and arachnoid, and downward as a thin tapering-off growth.

The growth had involved the 8th cervical and 1st and 2d dorsal posterior nerve roots on the left. These nerve roots were so intimately mingled with the tumor that they could not be liberated, and were of necessity divided during the removal of the growth. The anterior roots were left intact, the tumor being entirely behind them.

After the tumor had been removed the cord was lifted from the body of the vertebræ and thoroughly inspected anteriorly. No evidence of any further tumor could be found except for a small node about 3 mm. in diameter lying in a pit in the dura opposite the body of the 1st dorsal vertebra.

At the time of operation we were unable to determine the source of this tumor, whether an extension of the large tumor or an independent growth. The cord lying behind it was, by a thorough inspection, found to be free from tumor tissue. This was the only point at which the dura was involved in the lesion, and here the growth gave no evidence of having penetrated into the dura itself. The large tumor was removed without breaking it by dividing the arachnoid membrane and lifting it away from the cord. This was accomplished by sharp dissection aided by the use of moist cotton tampons. There was no laceration of the cord, and at no point was there any bleeding from the pia vessels. The small node in front of the cord was lifted out of its dural pit intact by means of a sharp bone curette. Careful inspection of the cord revealed no additional tumor tissue, and no evidence could be found that tumor tissue involved either the cord or the dura. No evidence of the central gliosis was obtained.

After removal of the tumor it was seen that the cord was pushed well to the right side of the canal and was compressed on the left lateral aspect to one-half or less of its normal diameter.

As presented to Dr. Elser, the "new growth was a soft and pliable tongue-like piece of tissue measuring $4\frac{1}{2}$ cm. in length and $1\frac{1}{4}$ cm. in breath and 4 mm. in thickness in the middle portion, tapering off toward the ends, which were less than 1 mm. in thickness. The tissue had a flesh-colored appearance

resembling that of the spleen. The outer surface was smooth and glistening, the under surface presented a coarsely granular appearance and the structure suggested that the growth consisted of congeries of bloodvessels which led to the tentative diagnosis of an angio-endothelioma. In general, it reminded one of a somewhat congested choroid plexus. Specimen No. 2 was a small piece of tissue, the size of a lentil, which was an isolated growth and macroscopically not connected with the main tumor. It was composed in part of blood-clot and in part of tumor tissue."

On the fourth day the evidence of operative damage to the cord began to subside, and by the seventh day power had returned in both lower extremities. The return of pain and temperature sense to the right half of the trunk and right lower extremity was complete—these having both been lost prior to operation. Muscular sense and sense of position had returned to the right lower extremity, but was noted on the twelfth day as absent in the left. The patient would carry out any muscular act requested with the left lower extremity, but unless she could see the part, had no knowledge that she had done so. Ankle-clonus was still slightly present and knee reflexes were exaggerated, but decreasingly so. The pupils were still somewhat contracted, and the left lid ptosis (sympathetic) was present. The preoperative root pain was present, but to a markedly decreasing degree.

The course of the disease after this date was marked by constant changes in symptoms, which were not explained on any basis of a change in organic lesion. For a period there seemed to be a slight but steady improvement in muscular power and paresthesia below the level of the 2d dorsal segment. Both lower extremities could be moved with considerable power in all muscles. At times there was, apparently, a correct appreciation of pain and temperature sense in the right body and lower extremity. Sphincteric action was normal. Reflexes exaggerated, abdominal absent, and the Babinski less marked. The contraction of the left pupil and the ptosis were less marked. The line of sensory disturbance on the body had gradually passed downward from the 2d dorsal to the 8th and 10th dorsal seg-

ments. On one occasion she experienced great tenderness in the postcervical region—5th and 6th vertebræ, and marked rigidity of neck. This lasted several days, but gradually wore away without being explained. So far as could be determined the root pains were growing less all the time, though on occasion she complained bitterly of them, sometimes on the right side, but most often on the left. There was little, if any, change in the left arm atrophy. Her general condition was fair, though she was poorly nourished and showed markedly the long, painful strain.

December 4: The picture is markedly changed in the past forty-eight hours. The paralysis of the lower extremities is complete and the reflexes are lost except for a slight knee-jerk. No sensation of any kind is present from the original line of hyperesthesia (2d dorsal) downward. The advance upward from the spines of the ilia (which was the line four days ago) has been progressive. The most probable explanation at the present time is that a recurrence of the tumor has taken place at the original site, with possibly other tumors at other sites. In any case there is every evidence of a complete transverse block of the cord at the 2d dorsal segment.

December 5: Temperature ranged very high, reaching 107.8. No changes in neurological examination that could be determined, though the delirium which the patient developed made observations inaccurate. Respiration became of the Cheyne-Stokes type, and later very shallow sighing in character. Death occurred at 10.30 P.M., caused by respiratory failure of bulbar origin.

Autopsy. Reported by Dr. Elser. Recent scar in median line of back extending from the 5th cervical spinous process to the 4th dorsal spinous process. Dissection of scar shows perfect union in deeper parts and no evidence of infection. The spinous processes and laminæ of the 6th and 7th cervical and of the 1st, 2d, 3d and 4th dorsal vertebræ are missing. The exposed dura is fairly tense in this region and shows a thickened band in the middle line, extending longitudinally for a distance of about 4 cm. This band measures about 5 mm. at its widest portion,

gradually tapering off at both ends, and in its most prominent portion is about 3 mm. in thickness. It consists of a fairly soft tissue red in color, and looks not unlike granulation tissue. Its resemblance to the original tumor is sufficiently striking to raise the question of recurrence or implantation. Upon opening the dura the cord appears as a soft, almost diffluent mass of medullary tissue involving a segment about 2 cm. in length. Above and below this point the cord is normal in consistence. The whole cord from the 2d cervical to its lower end was now exposed, and apart from small masses of altered blood found in the subdural space in the middle and lower dorsal region and the accumulation of a considerable amount of straw-colored fluid at the lower end the regions below the site of the operative field present nothing unusual. Spinal fluid was withdrawn from the lower dorsal region for bacteriological examination.

On the anterior aspect of the cord above the area of compression myelitis referred to above there is a flat almond-shaped tumor mass identical in appearance with the original tumor. In the recent state it measures about 1.2 cm. in length, 6 mm. across at its widest point and 3 mm. in thickness. It is not adherent to the dura and appears to be covered with arachnoid. Tissue similar in consistence, structure and appearance is found in the root canals surrounding the nerves as they emerge from the spinal canal.

Microscopical examination of this tissue and of the thickened band corresponding to the line of incision through the dura shows it to consist of tumor tissue.

Bacteriological examination of the spinal fluid yielded negative results. The cord was fixed in formalin and sent to Cornell for a more detailed examination.

The illustrations show very well the situation and the general appearance of the tumor as found at operation, and of the cord and the secondary tumor found at autopsy.

At autopsy the cord was seen to be practically in two pieces, the combined pressure of the two tumors having almost destroyed it about the level of the first dorsal segment. Lying approxi-

mately at the level of the original tumor, but on the left antero-lateral aspect of the cord, was a tumor measuring 2 cm. long, $1\frac{1}{4}$ cm. wide and $\frac{3}{4}$ cm. in thickness. This tumor was irregularly oval in shape and lay upon the cord and beneath the arachnoid. An anterior nerve root was stretched over this tumor and was adherent to it. The right lateral border of this tumor reached past the midline of the cord in front and seemed unattached to the cord at this place. The left lateral border of the tumor cannot be defined, for it fuses with the dura and with the broken-down cord as it curves around to the left and posteriorly. The tumor is grayish in color but darker than the cord itself. It is streaked here and there with reddish brown. It is firm to the touch, but a little softer than the normal cord. It occupied the exact site from which the small anterior tumor was removed.

The dura for a distance of about 7 cm. along the left lateral aspect of the cord, in the region of the tumor, is discolored, thickened and indurated. It is slightly adherent to the tumor in front. The inner surface of the dura here and there in the vicinity of the tumor is studded with smooth, lentil-shaped elevations resembling fat, but, as sections later proved, were not nearly so innocent. The only other notable feature of the cord before section was a number of small, very white, bony plaques in the arachnoid here and there at levels below the tumor, the largest being not more than $\frac{1}{2}$ cm. in diameter.

Sections from the original tumor are composed of cells in masses and strands separated here and there by delicate fibrils. The strands of cells are often split by wedges of hemorrhage. The secondary tumor and other local metastases are much more cellular in character, and fewer hemorrhagic areas are seen. The cells composing the tumors are round or oval and have a large light blue staining nucleus, usually round or oval in shape, with a well-defined granular nuclear membrane. Within the nucleus is a delicate but well-stained chromatic network. There is a round nucleolus which stains somewhat less distinctly than this network. Surrounding the nucleus is a scantly, ill-defined cell protoplasm, and between the cells can be seen, here and

there, delicate, ill-stained fibrils. These fibrils do not stain well with eosin and not at all by Mallory's phosphotungstic acid hematoxylin.

There is only occasionally seen a tendency to rosette formation.

On sectioning the cord above and below the site of the tumor there was found an oval pencil of what appeared to be hemorrhage running up and down the cord in the left posterior horn. This could be traced upward for about 1 inch and downward for about $1\frac{1}{2}$ inches from the destroyed portion of the cord. Microscopically it is composed of proliferated glia tissue forming the central portion of the oval, and hemorrhage forming an outer lamina about the gliosis. In the hemorrhagic zone can be seen numerous cells loaded with deeply staining pigment granules. In one place below the tumor, near the lower end of the pencil of hemorrhage, there was found a small, round area of rarefaction in the white matter in the right, cornucommisural zone of the posterior column. This appears to be another small focus of gliosis, but without associated hemorrhage, as in the case of the area within the left posterior horn just described. Secondary degeneration of both pyramidal tracts can be seen in sections of the cord below the tumor.

We have designated this tumor as a neuroepithelioma because it possesses the structure of such a tumor, and we believe this name most accurately describes its characteristics.

Most of the descriptions of neuroepithelioma refer to tumors of the suprarenal bodies or of the retina (so-called glioma retinæ). Only a few of these tumors discovered in the spinal cord or brain seem to have been described in the literature.

Neuroepithelioma may be taken to mean the same thing as neuroblastoma or neurocytoma, and, indeed, Ewing[7] describes neuroepithelioma as one of the three structural forms of glioma, stating that the "adult form of glioma consists of well-developed glia tissue, but in many embryonal tumors undifferentiated neural epithelium appears and the tumors are called neuroepithelioma." Although there is some objection to it, the term gliosarcoma has often been used to describe the very cellular

type of tumor composed of round or spindle glia cells deficient in fibrils.

Mallory[8] defines a neuroblastoma as "a tumor of epiblastic origin, of which the cells tend to differentiate into nerve cells."

Since the nerve cells in the central nervous system and outgrowths from it, like the eye, ear and the sympathetic system, differ much in appearance and function, it is to be expected that neuroblastomas originating in these various places will vary somewhat in appearance. For example, a neuroepithelioma arising from the internal granular layer of the retina is usually composed of cells which are smaller than those composing the suprarenal tumors, although otherwise the tumors are not unlike (Fig. 17). The constant tendency of these tumors, whether found in the brain, the cord, the retina, the medulla of the adrenal or elsewhere, is to form cells with a prolongation of the cytoplasm at some point into an axis-cylinder or nerve fiber. In addition these cells have a tendency to arrange themselves into little balls or spheres which, when cut through, give the appearance of "rosettes" with the neurofibrils running in tangled masses toward the center. If the tumor is not too rapidly growing and the cells have had time to become differentiated, then the fibrils are well developed, and can often be readily demonstrated by eosin in ordinary preparations stained by hematoxylin and eosin. Such examples of the tumor are less malignant than the more cellular types where the fibrils are few and may resist staining by eosin, and even the specific stains for neuroglia and neurofibrils. The tumor we are describing in this paper is of the latter type.

In addition to the sites of occurrence already referred to, neuroblastomas have been found to arise at the root of the lung, in the sacral region, in the region of the scapula (Symmers[9]), the inner side of the thigh, probably originating from the femoral sympathetic plexus (Ewing[10]) and in the retroperitoneal region. Symmers says "that the recognition of primary neuroblastomas in almost any situation in the body may be safely predicted is signified by the fact that undifferentiated nerve cells migrate

from the embryonal nervous system to form the nerves and ganglia wherever encountered."

It seems to be true, also, that the more embryonal type of tumor may develop within an ordinary glioma, as described by Uematsu[11] and others. Schlapp[6] believed that in his case the neuroepithelioma developed as a result of operation in a case of syringomyelia. In our case the more embryonal type of tumor was at least associated with gliosis of the cord, although the exact relationship between the two processes is not clear. The association of neuroepithelioma with ordinary glioma or gliosis is not surprising when we consider that the neuroglia has a common origin with the nerve cells and their processes from the cells of the neural groove.

The cases of neuroepithelioma of the cord and brain have been found in older people than is the case with the retinal, sacral and suprarenal tumors. Symmers' case in the scapular region was in a man, aged forty-four. Schlapp's case was in a man, aged forty-three. Our own case was in a woman, aged forty. Symmers' case and our own followed some time after an injury, although the relation of this to the subsequent tumor formation is no more clear than in most associations of this kind.

The tumors of the retina and suprarenal are very malignant, forming metastases in distant organs, such as the liver and brain. Our tumor is also a malignant one, forming local metastases and very rapidly growing, especially after operative interference. In this respect, at least, such tumors differ greatly from the ordinary kinds of glioma.

From our introductory paragraph it may be judged that we look upon neuroepithelioma as a tumor which develops from embryonal, undifferentiated nerve cells wherever these cells may wander to in course of the development of the central nervous system or its outgrowths. Many of these tumors in the retina, the suprarenals and the sacral region have been congenital, and, in the case of the retinal tumors, at least, are rarely found after the first few years of life. Wintersteiner[12] believes that in the case of glioma retinæ the tumor arises from the growth of misplaced elements of the outer granule layers.

The study of the cord and the tumor give our conception of the nature of the tumor. There are important considerations coördinating the pathological process with the clinical course. It is difficult not to associate the trauma with the development of the subsequent lesion. While there were intervals after the trauma when symptoms were not present, the patient connected the pain with the injury, and subsequent developments demonstrated that the lesion coincided closely with the site of the injury.

The tumor lay beneath the arachnoid. This may readily have been the seat of a pial hemorrhage and other damage from the injury. The reported cases of close association between trauma and cord tumor make it probable that injury to the nervous tissue is more prone to be the starting point of tumor formation than other tissues (Schlapp and Bovaird[13]).

The life history of the tumor is of interest in this connection. The injury occurred in November, 1919, and was followed by only a transitory inconvenience a few days later, apparently due to muscular and joint injury rather than any damage to nerve elements. Twenty months later the first pain, evidently due to nerve change, appeared, but was only present for one or two months, to be followed by complete cessation for a period of four or five months. After that there was some remission, but never complete relief, and nine months was required before evidence of cord compression appeared. If one connect the trauma with the tumor growth it is necessary to postulate a period of some months following the former before the cells lost their normal restraints and took on the characteristics of malignancy.

The incidence of the central gliosis found on section of the cord is a matter of conjecture. Certainly no symptoms referable to this lesion were present at the time of operation, unless one assumes that the hand atrophy was so caused. The gliosis lies too far posteriorly in the cord to make this probable.

The very confused picture developing in the seven weeks etween the operation and death may in some measure have

depended upon the cord destruction caused by this growth. One is forced to the conclusion that the gliosis did not antecede the formation of the neuroepithelioma, which is a reversal of the conclusions from the study of Schlapp. It is recognized that the injury may have produced a hematomyelia which in turn resulted in the gliosis. This view, however, does not seem substantiated by the facts given. A very practical question arises as to whether operative interference should have been earlier undertaken. The patient was under constant observation while the tumor was steadily increasing in size, with added damage to the cord, and yet no symptom developed which was incompatible with an inflammatory lesion of the nerve roots.

Simon,[14] Dana,[15] Laroche,[16] and Starr,[17] with many other writers, give ample evidence that the pain of a radiculitis or pachymeningitis may show all the characteristics that where present in this case. Frazier and Spiller[18] make this observation: "Given a case with pain of definite localization persisting without variation in its original territory for months, and especially for longer periods, one should at least have in mind the possibility of spinal tumor; but pain alone, even though localized for a considerable time, may be caused by radiculitis or meningomyelitis. Pain associated with atrophy in root distribution of the upper level should arouse the suspicion of tumor, and yet it seems to us, as a general rule, advisable to defer operation until some clinical evidence of implication of the cord is obtained."

Camus[19] writes extensively on radiculitis and quotes several authors showing the difficulty of assigning a definite pathology to this lesion. He says certain primary tumors of the spinal meninges at their inception by compression of the roots produce only the symptoms referred to the roots. At this period the diagnosis most often made is radiculitis. Camus states that the posterior roots are more susceptible than the anterior and that the pathology is an inflammation of the roots themselves.

Mills and Williams[20] comment on the impossibility of differentiating the two conditions under discussion. The pain is possibly more excruciating and sharper or more lancinating in

tumor cases, while those of pachymeningitis often have some rigidity and soreness of the neck and aching of the shoulders. The writer (J. A. H.) has observed 2 cases of radiculitis or neuritis in which the pain was quite as severe as that described in this case. In both there was the same atrophy of the hand muscles, and both continued uninterruptedly for a longer time than here elapsed before cord compression became apparent. Both recovered.

It seems evident that in the face of the testimony of so many observers a true diagnosis cannot be made until medullary symptoms appear. It is worthy of emphasis, however, that tumor formation in the posterior lateral region may be so closely related to the posterior nerve roots that root pain is the only symptom for considerable periods. The type of tumor here studied, because of its soft texture, its point of origin beneath the arachnoid, and its retention under this membrane, is particularly adapted to exerting the baneful pressure on the roots.

So far it seems impossible to predict the nature of these tumors in the cord or brain before operation. When found at operation they may have the most innocent appearance and be capable of rather easy removal. However, like the related tumors of the adrenal and retina, they are malignant, with a tendency to form metastases in the neighborhood, and, potentially at least, distant metastases as well. It would seem to be wise, therefore, to remove them with as little disturbance of the tumor as possible, and to realize that removal of any portion of them by curette or sharp dissection may be followed by recurrence of the tumor with a rapidly fatal result.

The question as to whether an earlier operation would have made recurrence less probable must be answered in the negative. At no time would removal have been accomplished with less trauma or greater certainty of removing all potential tumor element. The future must determine the final value of radiotherapy, but our present knowledge holds out at least a hope that these tumors are susceptible of destruction by radioactive agents.

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DISCUSSION ON THE PAPERS OF DR. BRUCE AND DRS. HARTWELL AND STEVENSON.

Dr. W. J. Mixter, Boston:

I wish to say a word in regard to the case reported by Dr. Stevenson. In the first place I feel that this is a very interesting type of tumor and must be classified as of extreme malignancy. The gliomas, both of the brain and cord, vary greatly in malignancy from the slowly growing invasive tumor without metastases at one end of the line to the type reported in this paper at the other.

Another point in this connection is the diagnosis. The diagnosis of this case was delayed, so Dr. Stevenson tells us. There was no mention made of a study of the spinal fluid. I feel absolutely sure that examination of the spinal fluid by lumbar puncture, or better by combined puncture of the cisterna magna and the lumbar space, is of the greatest importance in the early diagnosis of such a lesion. Cord compression can almost certainly be demonstrated by that measure, thus ruling out the degenerative lesions, a neuritis or possible multiple sclerosis. In the last 25 cases of cord compression which have been under my care we have carried out this procedure as a routine, and it has only led us astray once in that number of cases, and that was a very flat dural cyst. There have been 5 cases in that group which showed no symptoms of cord compression by lumbar or combined puncture, but which were operated upon by me on account of the clinical symptoms, and in none of these 5 cases was I able to find any tumor. As time goes on, and I feel more certain of the accuracy of this procedure, I find I am operating on a smaller number of cases that result in a negative exploration.

Dr. Stevenson (in closing):

In closing, I should like to say just a word. As a matter of fact, if Dr. Ayer's procedure had been carried out the diagnosis could probably have been made earlier. Unfortunately, this was one of those cases in which it is sometimes impossible to do even a lumbar puncture until it is too late. When lumbar puncture was done the fluid was xanthochromic. We knew then that we were dealing with a cord tumor. There were only five cells to the cubic centimeter, and increase in the globulin and everything else was negative. I do not think we can diagnose such a tumor as this before operation, but I think the point is that if we do diagnose it on section after operation the only chance seems to be to use some kind of radiation, because if

a tumor like this is removed even without any trauma at all, which seems inconceivable, it will rapidly spread and cause complete destruction of the cord or form metastases elsewhere. I think the only way we can differentiate between the malignant and non-malignant types is by the presence or absence of neurofibrils.

The last slide I showed was from a tumor that had existed at least seven years, had no metastases and was operated on without difficulty after that lapse of time.

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